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Table of Contents.

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ORIGINAL ARTICLES— PAGE.
The Sir Richard Stawell Oration, by T. P. DUNHILL 139
Frequency of Micturition in the Female, by W. JOHN CLOSE, M.B., M.S., F.R.C.S., F.R.A.C.S. .. 147
An Operation for Thenar Paralysis, by N. D. ROYLE, M.D., Ch.M., F.R.A.C.S. 155

REPORTS OF CASES—
Extradural Abscess Secondary to a Carbuncle of the Wrist, by M. E. CHINNER, M.B., B.S. 157

REVIEWS—
Cardiac Output 157
Progress in Dermatology 158
Hygiene and Public Health 158
History of Tuberculosis: A Correction 158

LEADING ARTICLES—
The Adelaide Congress 159

CURRENT COMMENT—
Punch-Drunk 160
Epidemic Myalgia 160
Research in Tropical Medicine 161
The Sir Richard Stawell Oration 161
The Australian and New Zealand Association for the Advancement of Science 161

ABSTRACTS FROM CURRENT MEDICAL LITERATURE—
Ophthalmology 162
Oto-Rhino-Laryngology 163

SPECIAL ARTICLE—
Virus Diseases: The Present Position 164

BRITISH MEDICAL ASSOCIATION NEWS— PAGE.
Scientific 167
Nominations and Elections 168

MEDICAL SOCIETIES—
The Public Medical Officers' Association of Victoria 168

CONGRESSES—
The Australian and New Zealand Association for the Advancement of Science 168

RESEARCH—
Fellowships and Appointments for Research in Tropical Medicine 168

CORRESPONDENCE—
The Law of the Split Pelvis 169

OBITUARY—
Danial Florance MacGillicuddy 169
John Reginald Davis 169
John Smith Purdy 169

CORRIGENDUM 169

NOTICE 169

BOOKS RECEIVED 170

DIARY FOR THE MONTH 170

MEDICAL APPOINTMENTS 170

MEDICAL APPOINTMENTS VACANT, ETC. 170

MEDICAL APPOINTMENTS: IMPORTANT NOTICE 170

EDITORIAL NOTICES 170

The Sir Richard Stawell Oration.[1]

By T. P. DUNHILL,
London.

I DESIRE to express my deep appreciation of the honour the trustees have conferred upon me in asking me to deliver this lecture. I also want to give my personal thanks to Dr. A. E. Rowden White for his foresight in instituting a lecture to perpetuate the memory of one whom this generation would wish to honour.

I had always believed that I should absolutely decline to undertake a commitment of this nature; but it was not possible to refuse my tribute to one who, at the time I was invited to do so, was alive and my very dear friend. When your trustees, realizing something of the length and depth of that friendship, and knowing that after an absence of fifteen years I was to visit Melbourne, thought it fitting to ask me, a refusal was not to be contemplated.

It is a great grief that I can never have the pleasure I had anticipated in expressing to him my own and your affection and admiration. The possibility that he might not be here had not crossed my mind. I had never known a Melbourne without him and had taken it for granted that we might continue to work and play together for a time to which it had not occurred to me to put a period. At the beginning of this address I would note my desire, which would most emphatically have been his, that this should be no sad memorial. That would be foreign to the man, for to Richard Stawell life was work, service, joy; always contribution—to his family, to his friends and to the wider public. This impulse has created a wonderful legacy which we have inherited. We are indeed filled with pride in the man and gratitude for the friendship we

[1] Delivered at Melbourne on September 4, 1935.

have enjoyed. I am not unmindful that a family has lost a husband and father and that you have lost a friend. My trip to Australia has been robbed of the one expected joy that stood out above all others; but it has been a rare privilege that Sir Richard Stawell lived in our lifetime, and to such purpose that for generations men will be the richer.

In earliest recorded history two vocations were open to the human race—hunting and tilling the soil. Life soon ceased to be so simple, and it was not long before the hewing of wood and the drawing of water were whole-time jobs. In Grecian and Roman times, and long before, life had become complex, with specialists in ever-multiplying departments. Cicero chooses a barrister's career, Hippocrates becomes a physician, and so to our own times. In every profession men are ambitious and are in competition with their colleagues. Ambition may have various goals: scientific achievement, social success, the financial rewards that sweeten toil; but I wonder if in any calling other than medicine unselfishness has existed side by side with accomplishment to an equal degree. Readers of Robert Louis Stevenson, remembering the preface to "Virginibus Puerisque", know that he had no two minds about this; and George Moore, when he dedicated "Aphrodite in Aulis" to Sir John Thomson-Walker, spoke with equal conviction. And yet it is certain individuals who have raised a profession to this high vocation. Everyone who has stayed the course and passed the examinations has seen much to make him think. As John Brown said of his Scottish hills, "they rectify and abate a man, if he is worth the process"; but the man's the man for a' that; and in spite of those rectifying processes there are many who, though worthy and kindly, fail to reach full stature. We know men who have achieved considerable social success; others again who are able and full of courage, but not devoid of selfishness; some of us may be knowledgeable, dependable, but reticent and lacking some subtle sympathy with our fellows. But all who knew Stawell well will agree that he was first a father to us, then a friend and brother—an affectionate chum—filled with knowledge and wisdom and patience and tact: knowledge, that has been our admiration rather than our envy, for he so easily kept ahead of us; wisdom, which is denied to many who have knowledge; patience, heavily tried in the classroom, for not all students are heaven-born or heaven-sent; and tact, enabling him to temper sarcasm with such kindliness that, while we smarted under the merited rebuke, the facts sunk in and the kindliness prevented any sting.

It has been given to few to be able to convey so clearly to the minds and hands of untrained youth in an out-patient department the methods of physical examination, the ability to elicit signs, to sift, marshal and interpret them. Then as we who were the youths became less "untrained" and followed him to the wards, it grew upon us that he never passed a bed containing a patient whose symptoms could not, in the present state of knowledge, be interpreted. To those of you who are not medical it may come as news that there are illnesses whose diagnosis still defeats us! And those of us who are medical know how convincingly a teacher can occupy himself on the other side of the ward so as to pass such a bed, lest his students might have some doubts of his omniscience. Those who lived a few generations ago know how skilfully Achilles learned to hide his heel. There Dick Stawell was like Mark Tapley, for he "came out strong", and beside such a bed we were made to face the patient and his difficulties, to think for ourselves and use whatever intelligence we possessed. That inborn honesty combined with that inborn courage made him unable to shirk any responsibility; and if we had it in us, it made it impossible for us ever afterwards to ignore a difficulty and remain comfortable. Stawell and Maudsley started a new era in clinical teaching, and it is due to those two that a generation of teachers has arisen in your hospitals and university, which is turning out students who are holding their own in any community in the world.

The record of Sir Richard Stawell's family, his early life, his medical training, his achievements, were ably presented to you last year by Dr. Blackburn. I want to add only one word. Family traditions in our lands remain patriarchal—father to son—but, having known personally the mother of Sir Richard Stawell, I can say most emphatically whence he derived some of that mental luminosity which gave such grace—and occasionally a very kindly satire—to his great abilities. And one word about another, who joined him for life. We all wished the very best for our friend, but none of us could have hoped that anyone would shed such a radiance over his life and be in truth such a helpmate, and not only in household and domestic matters. At the appropriate times she would send him off with his men friends to snow-fields and inaccessible trout streams. In this way he constantly renewed his youth, for Dick Stawell never grew old. For that let us be grateful, for him and for ourselves, to Lady Evelyn Stawell.

The subject of this memorial lecture was settled many months ago by Sir Richard himself with the trustees of the oration. There are those who, inquiring and imaginative, choose subjects more or less philosophical, aiming to analyse the past and the present, and possibly illuminate the future. I am not of that company, but of those who are happier when speaking of more practical matters. With that limitation in view the subject of "Diaphragmatic Hernia" was chosen.

Diaphragmatic hernia, when not of traumatic origin, is frequently congenital. In many cases it is fatal in the first few hours or days of life; in others greater or less disability persists, and in some it is unsuspected until late in life.

The condition is still rare. Even recent text-books published in the United Kingdom scarcely mention

it, except when due to trauma, but it begins to appear more frequently in our medical journals, usually with the account of a single case. Dr. A. F. Hurst, known to most of you, has reported a number of cases in the *Guy's Hospital Reports*. Woodburn Morison has written on the radiological aspect in *The Proceedings of the Royal Society of Medicine*. My friend R. P. Rowlands, recently deceased, performed, I believe, the first successful operation for the condition in England. Barrett and Wheaton have written on diaphragmatic hernia in children. I know that H. Douglas Stephens, W. Hailes and others in Australia have performed successful operations. If I have omitted names of my countrymen or details of their work, it is only because you have not made your own work available in the literature.[1]

In America, as you know, Hedblom wrote on this subject many years ago. A little more recently Stuart Harrington, from the Mayo Clinic, has reported a large series of cases. It had puzzled me why, until a few years ago, so few cases were recorded in the United Kingdom. I believe the only reason why so many have been found in America is because in those large clinics the examination of patients is more complete. The recognition of rare conditions and many of the advances in medicine and surgery are achieved through the routine of careful and detailed examination of every patient. If we do not realize the possible existence of a pathological condition, naturally we do not find it; and some patients continue to suffer from "neurasthenia" only because the cause of their pain is not discovered by the ordinary methods of examination.

I saw my first patient with diaphragmatic hernia in 1924. The patient was under the care of Dr. Chittenden Bridges and Dr. Fawcett, and the X ray examination had been made by Dr. Lindsay Locke. From that time, looking carefully with my colleagues into the cases of patients who were suffering, but in whom the diagnosis was not obvious, thirty-seven cases have been found. In seventeen of these the œsophagus has been of normal length, one or more of the abdominal organs being herniated through an opening in the diaphragm. In the others the œsophagus was short and part of the stomach had developed in the thoracic cavity. This thoracic loculus becomes progressively larger, being pushed by abdominal pressure and drawn by inspiratory suction "upstairs". It is clothed with peritoneum and is surrounded with a peritoneal sac.

The classification of diaphragmatic hernia is not yet very satisfactory, and the reason is that so few have been seen and examined in the *post mortem* department. The large defect that we see when a patient finally reaches operation has obscured what was probably its small origin, and at operation time is of such importance that we cannot linger to make minute dissections. Each succeeding operation case helps us to interpret more accurately what we think we have seen and to confirm or otherwise what we have suspected in previous cases. So, little by little, but very slowly, our knowledge grows.

I have been greatly helped by Professor Woollard, well known to all but the youngest of you, a distinguished graduate of this university and now Professor of Anatomy at Saint Bartholomew's Hospital;[1] but he has not had a fair chance. His contact with the patient is restricted to observation of the field while the operation is being carried out, and this in only a small number of the cases. What he can see in these circumstances does not give him the same opportunity as would be given by leisurely dissection of the cadaver. We discuss the radiograms with him beforehand, and, using these, his knowledge of embryology and anatomy, such view as he can get of the operation field and our description of what we believe we are handling, we hope that we are learning. It is scarcely possible as yet to propound a universally acceptable classification.

I have classified my cases as follows:

	Cases.
1. Retrosternal (synonyms: parasternal, foramen of Morgagni)	1
2. Left dome	4
3. Costo-vertebral region, left (synonym: *hiatus pleuro-peritonealis*). (?) Non-development of left crus	4
4. Œsophageal region:	
(*a*) *Hernia diaphragmatica transversa*. The œsophagus is of normal length; the anatomical hiatus is replaced by large defect. (?) Due to non-development of crura	3
(*b*) Para-œsophageal. The œsophagus is of normal length; the hiatus is anatomically normal but dilated	5
(*c*) Short œsophagus, with partial or complete thoracic stomach	20

I hope that the classifications employed by different men will gradually approach uniformity and that they will be kept as simple as possible, slight variations being described as such but kept under their main subdivisions. Already I begin to doubt if some of my own descriptions are accurate.

1. The retrosternal type is definite. As well as my own case of this, others have been published, and I know one museum specimen.

2. Through the left dome. These cases were so clear that the situation and character were undoubted.

3. The anatomy of the type occurring in the costo-vertebral angle, hitherto called pleuro-peritoneal, is more doubtful. It may be, as is usually stated, that it occurs in the site of the original pleuro-peritoneal hiatus; but a defect in development of a crus might permit enlargement of the hiatus, which, if it became very large, could allow the development of a para-œsophageal hernia. This might reach the size of those described as costo-vertebral and illustrated in Figures I, II and III.

[1] Since this lecture was delivered, H. Douglas Stephens, W. Hailes, H. K. Pavey and Ian Hamilton have published excellent accounts of their cases in *The Australian and New Zealand Journal of Surgery*, October, 1935.

[1] Recently appointed to the Chair of Anatomy tenable at University College Hospital.

When I published my last paper[1] I had had three in this situation, all on the left side. Since that time I have operated upon a very large right-sided one and have seen excellent radiograms of another also situated on the right side.

Regarding 4 (*b*), I have had five more of this type since my previous paper, some of them very large, and, as I have just stated, I cannot help wondering whether this type merges into Type 3. Professor Woollard has pointed out to me that the muscularization of the diaphragm begins at two points (Figure IV), one in either side of the developing diaphragm. From these two points the

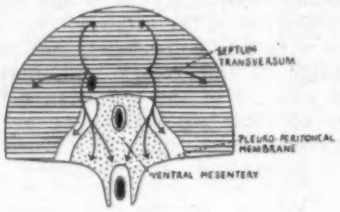

Figure IV.
Showing where muscularization commences and the direction of spread.

process spreads (Figure V), the crura developing last. I begin to wonder if muscularization may occasionally fail to reach its insertion, one or both of the crura remaining undeveloped. If both remained undeveloped, the condition to be described under 4 (*a*) would be explained (Figures XIX and XX). If one only failed to muscularize completely, it might possibly explain Number 3 in the classification table. A small defect in the attachment of a muscular structure in fœtal life may ultimately allow the margins of the defect to spread so widely that it becomes almost impossible, or very difficult, to determine the site or cause of the original defect.

Let us return to our classification, in which the cases are grouped according to the site at which

Figure V.
Muscularization more advanced.

radiography and inspection, and to some extent traditional nomenclature, lead us to place the defect. Figures VI and VII show an example of retrosternal hernia. In a man, aged twenty-four years, the termination of the ileum, the caecum with the appendix, and what should be the ascending and transverse colon are seen within the thoracic cavity. The colon is then seen to leave the thorax and descend almost directly to the left iliac fossa. The entrance and exit show that the neck of the sac is fairly small; the fan-shaped spread of the herniated structures in the thorax show that the peritoneal sac is large, extending from the right chest wall to beyond the mid-line, and high in the thorax. The radiologist (Dr. Simon) has obtained a lateral view with both the hernial contents and the œsophagus filled with barium, and from this we know that the œsophagus is of normal length, that it enters the abdomen in its normal situation, and that the posterior margin of the hernia is as definite as the lateral margins. We therefore know fairly accurately before the

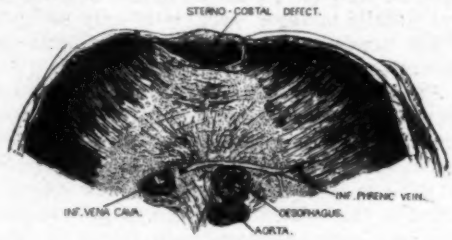

Figure VIII.
Drawing to show retrosternal defect in diaphragm.

operation is begun the degree of integrity of the remainder of the diaphragm, and which organs are or are not in the hernia. It is important in these cases, as in all others, to collect every possible scrap of information before operation, for the operation in some cases must be lengthy and the patient's life may well depend on its not being too prolonged.

The hernia illustrated in Figures VI and VII almost certainly began before the rotation of the colon occurred in fœtal life. Figure VIII is a drawing to show the situation of the defect from the under-surface of the diaphragm.

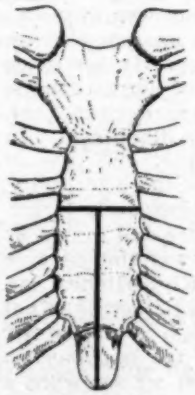

Figure IX.
Lines of section of the sternum.

It is, I think, more appropriate to speak of the operative procedure after each type rather than group all operations at the end.

In planning the approach for this case it did not seem possible to deal with it completely from the abdomen. The incision was therefore started in the mid-line of the epigastrium and continued upwards over the lower half of the sternum, which was then split and its leaves retracted (Figure IX). This gave good access; the viscera were returned to the abdomen; the sac, which was lightly, though firmly, attached throughout its whole extent, was gently peeled off and cut away. The defect was, in its posterior half, easily closed with catgut sutures; in its anterior half, by fascial graft sutures.

There is one interesting point. A radiogram taken before this patient left the hospital showed that the caecum, which at operation had been sutured to the abdominal wall just below the liver, had migrated to the right iliac fossa (Figure X).

It had never lost its urge to finish its fœtal journey, interrupted by hernia formation before the rotation of the bowel had been completed.

In the second type of diaphragmatic hernia the defect is through the left dome. Figure XI is a drawing showing the site and appearance from below, which helps to interpret the condition shown in the radiograms. The defect in the case to be described is not in the situation of any natural opening in the diaphragm.

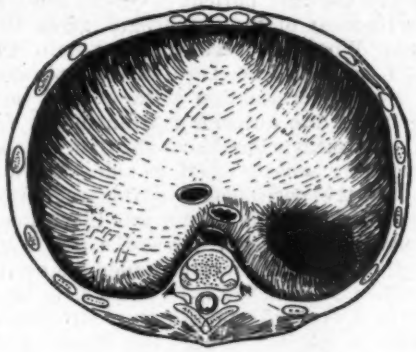

FIGURE XI.
Drawing from below to show defect in left half of diaphragm.

It occurred in a woman of fifty-two years, in whom the symptoms had only existed for two or three months. The margins consisted of thick muscle and there was no peritoneal sac. All these features make one doubt the congenital origin of this hernia and suspect a localized trauma or infection with rupture due to some strain.

Figure XII shows the œsophagus in this case to be of normal length, the stomach entering the thorax, a small portion of it containing barium, the remainder greatly dilated and containing gas, almost filling the left thorax and pushing the heart to the right.

Figure XIII shows the entering cardiac end and returning pyloric end of the stomach, made clear by reference to Figure XIV, drawn from the condition found at operation. At operation it was found that no sac was present; the stomach was almost completely within the left thorax, enormously dilated, fixed by firm adhesions to the base of the lung, which was crowded into the apex of the thorax and densely adherent around the margins of the defect. The patient was living in great distress.

This case seemed to necessitate a double approach—through the thorax as well as the abdomen. The thoracic adhesions could not be separated from the abdomen, nor could those around the margins of the defect be safely dealt with from above. A stomach of this size could not be returned to the abdomen without deflation, nor could it remain comfortably in an abdomen long unaccustomed to house it without the institution of some temporary means of deflation. A gastrostomy was therefore performed and a rubber catheter left in until the patient became comfortable. This was not done to give food, but to allow exit for gas.

A second patient, a child, with a hernia of this type has not yet been operated upon. He has attacks of pain from time to time, but when the attacks pass his mother wishes to delay.

In Type 3 the hernia occurs through the costo-vertebral angle. It has usually been called hernia through the pleuro-peritoneal hiatus. I have had three cases on the left side, one on the right. In all of these both the hernia and the defect were very large. In two the stomach reached the sterno-clavicular articulation. In each the stomach was rotated on its axis (Figures II and III). The transverse colon was also drawn up into the thorax. The pain simulates that of gall-stones, but is of more "bursting" character, generally, but not always, associated with the ingestion of food. One patient stated: "I look at food, I want it; I do not take it." This woman has since operation given a dinner to the staff of the nursing home each year on the anniversary of her operation.

In another patient hæmatemesis and melena were severe. At operation the defect was found to extend to the œsophagus, this structure being quite obvious under the peritoneum throughout its lower few inches to its junction with the stomach. (It is this, taken in conjunction with Type 4 (*a*), which makes me suspect failure of the diaphragm completely to reach its insertion, and that this failure, like a hare lip, permits muscular pull to separate the defect increasingly widely.) The size of the defect and the poorness in "holding" quality of the fibrous and areolar tissues around the œsophagus

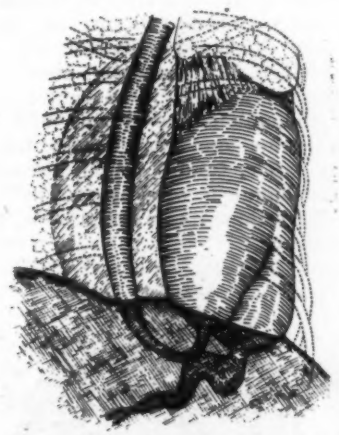

FIGURE XIV.
This drawing explains Figure XIII. It shows the adhesions between the stomach and the compressed lung. It is an exact reproduction of another film (with the addition of the adhesions drawn in).

and in the region of the vertebra make it difficult adequately to strengthen the repair. The repaired diaphragm may rise to a level higher than normal, as shown in Figure XV, but the stomach remains below the diaphragm; its axis is in the normal direction and the functional result is completely good.

Hernia of this type on the right side is rarer. Figure XVI is a radiogram of the condition in a girl aged twenty upon whom I operated in 1935. Figure XVII is a radiogram taken after operation,

showing the stomach in its normal position below the left dome of the diaphragm.

The fourth type is really a group including three types. I have had these drawn on one print (Figure XVIII) so that they may be seen together for comparison. The first of these, 4 (*a*), is the *hernia*

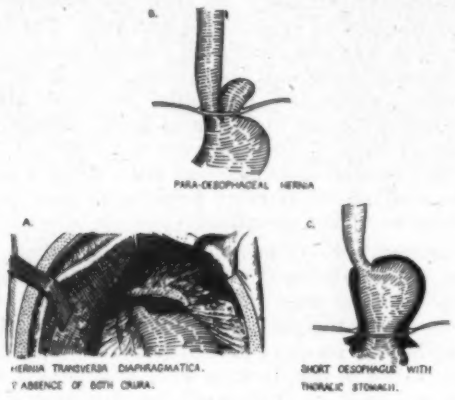

FIGURE XVIII.
Showing three types of hernia in the œsophageal area.

transversa diaphragmatica of the literature. The defect crosses the mid-line, is bilaterally symmetrical and of reniform shape, the concavity being caused by the vertebral column; the convexity is caused by the thickened posterior margin of the diaphragm which is apparently devoid of crura. The peritoneum, having no muscular support, is pushed by abdominal compression and sucked by inspiration higher and higher into the posterior mediastinum. The fundus of the stomach follows it. The œsophagus is of normal length (Figures XIX and XX), but its lower end is carried up with the stomach.

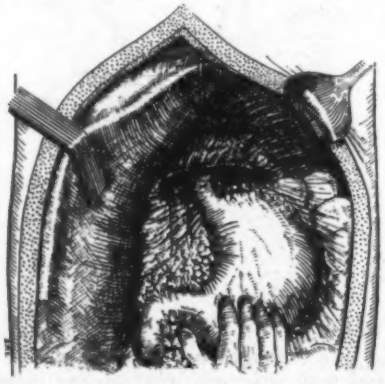

FIGURE XIX.
Drawing to show transverse diaphragmatic hernia in which the stomach slides up and, unless held down, occupies a position in the posterior mediastinum, as shown in Figure XXI.

A woman, aged fifty-four years, suffering from this type of hernia, was related to one of my younger colleagues. She had carried on for years until life began to be very difficult. Dyspnœa was troublesome and a feeling of tightness and pain was always present across the lower sternal region. Turning in bed caused such discomfort that she had developed the habit of sitting up, turning and then lying down again. This hernia was approached by the abdominal route. The left coronary ligament was divided and the left lobe of the liver turned back; only the pyloric end of the stomach was visible. The stomach was easily withdrawn and the appearance then seen is shown in Figure XIX. When the hold was released the stomach immediately disappeared up into the sac, where it had become accustomed to lie (Figures XX and XXI).

This was the first patient in whom the theory of the development of the diaphragm, given to me by Professor Woollard, seemed to explain the condition. I could find no crura. The diaphragm came to an abrupt termination with a thick muscular edge, forming the anterior margin of the reniform defect, this measuring about five inches laterally and extending equally to either side. The muscular defect left the peritoneum in this region without any "backing" and the hernia was inevitable. When the stomach was drawn down, the œsophagus and the cardia were quite obvious under the peritoneum, as shown in Figure XIX. I cannot help suspecting that Type 3 (costo-vertebral, or pleuro-pericardial) may have a similar cause, the defect being unilateral—usually left-sided, but occasionally right-sided—for in these the œsophagus and cardia are seen (from the thoracic aspect) simply covered by areolar tissue, pleura and peritoneum. That again may raise the question of the para-œsophageal type, next to be described, for in that type the œsophagus is obviously immediately beneath the peritoneum, but in the para-œsophageal type, when large, the postero-lateral margin is very thick and is seen reaching its attachment to the vertebral column. In this (para-œsophageal) type a crus is present, but instead of being firmly attached to the vertebral column throughout its whole length, and therefore vertical, it is seen to be attached at its lower extremity and is then directed obliquely outwards and forwards, leaving the neck of the hernia on its medial side, the dome of the diaphragm arising from its left or outer side.

The second type of the fourth group, 4 (*b*), is the para-œsophageal, one aspect of which we have just been discussing. I have had five more cases of this type since delivering the "Arris and Gale Lecture" in 1934.

Here again the œsophagus is of normal length. The drawing (Figure XXII) shows the manner in which the hernia probably begins. Radiographically it is not easy to demonstrate the full-length œsophagus in a late case with a large thoracic loculus, for this loculus is so broad that it covers the last few inches of the œsophagus and is in such close contact with it that it is difficult to obtain a view of the œsophagus clearly demarcated from the stomach, even from a tangential position. However, a characteristic picture can always be obtained. The œsophagus, outlined with barium, is seen descending vertically, then abruptly deflected, forming an angle, and after that again descending

as a flattened, narrowed structure beside the loculus (see Figure XXIII). The shadow of its lowest portion is covered by the stomach, so that a picture comparable to Figure XX [Type 4 (*a*)] is rarely seen. At operation upon patients with this type of hernia I have used the abdominal approach. The site is deeply situated. The patients always appear to be stout. This is probably because they are hampered in their activities by pain and discomfort, and when other pleasures fail us we tend to make up for them by the enjoyment of food. This pleasure may remain even though it exacts some retribution.

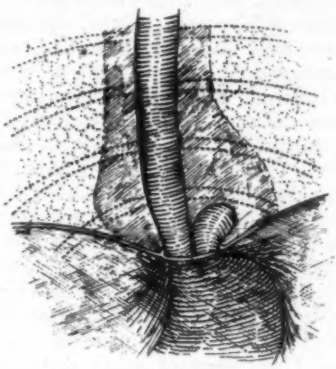

FIGURE XXII.
Initial stage in formation of para-œsophageal hernia.

Even if the diet has been restricted to that used for peptic ulcer, consisting chiefly of milk, and taken frequently, it is astonishing how much weight is gained when exercise is eliminated. Moreover, many of these patients are middle-aged, when even those of us who are normal easily acquire weight. When the left lobe of the liver is retracted and the stomach withdrawn the appearance is characteristic. A deep concavity, narrow—compared with the size of the sac—at its neck, and lined with glistening peritoneum, is seen. (Peritoneum always glistens, but with a spotlight directed onto it the sac seems more than usually polished.) It is bounded on its right by the cardia and œsophagus, and on its left by a thick muscular band which extends from the vertebra behind the stomach obliquely outwards, dividing two concavities, one the hernial sac, the other the (now smaller) dome of the left diaphragm, with the spleen nestling, almost hidden, close to the ridge—formed by the thick muscular band—which I take to be the remains of the left crus of the diaphragm, for it obviously supports the left dome on its medial side.

Very long instruments are required for the operation when the patient is short and stout and the ribs are rigid. I begin to think that, even in this type, when the sac is large the thoracic approach would permit a more secure repair. X ray examination shows that the anatomical result is not always perfect. One woman was seventy-seven years of age when she came for operation. She is now eighty-three and enjoys life thoroughly.

There is one patient whom I had placed in this class, but who, on further consideration, I think should be classed under Type 2 because of the absence of sac, the dense adhesions in the thorax and because there was some muscular material to the left of the œsophagus which enabled me to close the defect firmly. Notwithstanding this I lost the patient. For twenty years she had not been able to go out to dinner. The stomach was completely in the thorax, even the pylorus being two inches above the diaphragm (Figure XXIV). Adhesions fixed it so firmly and so universally that, working from the abdomen and thorax alternately, the operation took a long time. It finished up well, but the prolonged anæsthesia, with chest and abdomen open, caused a good deal of shock with shallow respiration. Some bronchopneumonia followed, with a small lung abscess on the right (unopened) side. The patient died one month after operation. A subsequent examination showed that the anatomical condition was perfect, the axis of the stomach normal, and the repair in the diaphragm could scarcely be found.

Possibly an oxygen tent would have saved this patient, because the trouble in her case was imperfect respiration following operation.

Finally, Type 4 (*c*), the short œsophagus type. I believe that Bailey was the first to describe this. Akerlund and others have since written extensively about it. Figure XXV shows the condition, the œsophagus terminating well above the diaphragm, the cardia and a loculus of the stomach clothed with peritoneum occupying the thorax.

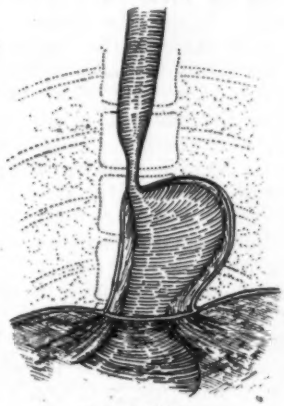

FIGURE XXV.
Drawing to show congenital short œsophagus.

In the earlier days the patient was usually sent with a diagnosis of carcinoma of the œsophagus, as shown in Figure XXVI. In some, a larger loculus of stomach ultimately rises above the diaphragm, and occasionally the whole stomach becomes a thoracic organ. Thoracic suction and abdominal pressure each tends to bring this about, and as more of the stomach rises rotation begins. Symptoms due to this condition vary. In some patients stenosis is present and the chief symptom is then dysphagia. There would seem to be some imperfection in development apart from or associated with the shortness of the œsophagus which renders the cardia more prone to stenosis. Dilatation occurs as shown in Figure XXVII, and ulceration is prone to follow this, as shown in

Figures XXVIII and XXIX. Ulceration may indeed precede the stenosis, as suggested by Hurst.

When ulceration occurs, pain in the substernal region and along the left costal margin is present. Hurst has shown that ulceration may also take place in the thoracic loculus of the stomach. Some patients have no dysphagia, but suffer from severe attacks of thoracic oppression. This is probably due to the thoracic loculus of the stomach becoming distended with gas and pressing on the pericardium and heart. Others suffer severe attacks of pain, due, I believe, to the thoracic loculus of the stomach becoming kinked over the margin of the diaphragm and being unable to empty easily. Some also suffer from attacks simulating asthma, but always following the ingestion of food.

Radiological technique has advanced greatly in recent years, and diagnosis becomes more accurate and more complete. This is a subject in which physician, surgeon and radiologist collaborate. The surgeon should see the X ray examination made and should elicit the points which are important to him—and incidentally to the patient. Œsophagoscopy is most helpful, in that it shows the local condition.

Treatment.—When dysphagia is the chief symptom, relief can be given by dilatation with the mercury bougie. It may be wise to use dilatable bags under vision through the œsophagoscope first, but in most instances this is unnecessary. In one case, in which the thoracic loculus became angulated over the margin of the diaphragm, I opened the thorax, crushed the phrenic nerve over the pericardium (which enabled the diaphragm to be manipulated with greater ease), divided this structure up to the œsophagus, transplanted the œsophagus laterally, and resutured the diaphragm around it almost, but not quite, above the cardia. This was only done in January, 1936, so I cannot yet speak of the late results.

I have not here dealt fully with the subject of diaphragmatic hernia. This is merely a record of some cases in a field not yet completely explored. It is meant to be stimulating and provocative, a tribute to my friend, who gave his consent to the choice of this subject when we all thought he would be with us.

And now to return for a moment to him in whose memory we meet tonight.

I have spoken of his work and a little of some work to which his example provided a direct stimulus. But while work is a man's job in life—is, indeed, its only satisfactory basis—it does not round off and complete his life. The soul of man is developed and revealed in his leisure; and of leisure modern life leaves us only too little—to our infinite impoverishment. The demands on Stawell's life left less leisure than remains to most men; and of that little the greater part naturally was devoted to his family, to reading and meditation, to music and art. Even though little remained, many of us were privileged to share in it. Our time chiefly came during those trips, to which I have already referred, to mountains and to rivers. In those days, or hours, or moments it was as though a veil was lifted and an ampler world—almost a magic world—was revealed to us. One experience, of which I have written elsewhere: winter on Mount Kosciusko; a nine-mile tramp through the snow on our skis to Bett's Camp; up hill all the way, metaphorically where it was not so actually. He began to tell me the story, and the deeper meaning, of Wagner's "Ring". You may have grown up with it; I had not. The intricacies of the *Rhinegold*, the Rhine maidens, the *Nibelungen*, right through to the *Götterdämmerung*. And as he talked one felt why at a performance of "The Ring" in Germany the audience is in tears; for they feel the gods struggling for existence in that primeval—or mythological—world and falling one by one, and Valhalla going up in flames. I was held rapt, for time had ceased to exist, and the miles, and the hills and the snow—no, not the snow, but the difficulties of the snow—were past.

Did that mean that he was a highbrow or a visionary? Not on your life! Take another day, again on Mount Kosciusko and also winter. A party of us; snow everywhere, but sun and blue sky over all; a group of rocks jutting out high through the snow; he, balanced on the highest pinnacle of the rocks on one foot, arms extended, telling the merriest and most ludicrous of stories, the cheeriest of a jolly party.

Was he practical, with all this? Another scene: The Yarra flooded; the rapids between Warrandyte and Heidelberg could only be negotiated in Canadian canoes at the time of the spring floods; and, whether you know it or not, it is then some river! The usual rocks and rapids disappear; islands materialize which normally are part of the banks, and trees stick out through the floods in a most disconcerting fashion. He and Charles Martin and Throsby Bridges had taught us something of the art of shooting the rapids, but we were repeatedly capsized. The canoes often—perhaps generally—received some damage. If we were lucky and could keep one hand on the canoe or a paddle, we might gradually be brought more or less safely to land, but not always on the right side of the river. If we were unlucky, crew, canoe and possessions parted company at once and were swiftly separated by the momentum of the flood. A sort of middle predicament occurred when the canoe was swept away from us, but we managed to grip the overhanging branches of a tree in midstream and, as we hung, reconnoitre and work out some way of reaching shore. And this was by no means easy. Rescuing an odd member of the party—there were always two, and sometimes three, canoes, each with two occupants—salvage of the canoe and its contents, building the fire, food, and reembarkation had all been standardized by practice, trial and error by Dr. Stawell. Here, as elsewhere, he was our father as well as our chum.

Spring time gave us the flooded river which we needed. It gave us something more—blossom time. The wattle trees overhung the river all along—silvery branches with golden blossom; and, a little later, peach orchards on the banks. There were other sorts, but, looking up from the canoes, the peach blossom seemed to make the air luminous and filled his soul with especial joy. Canoeing in high flood was courage with adventure. He had courage where there was no adventure—but a strong probability of reprimand. The following incident was a personal communication to me and is the only episode I have mentioned in which I was not actually concerned. An Australian hospital on a bleak island. Medical officers, staff and nurses drawn from a civilian population; no amenities, no comforts, few enough necessities. Illness—and severe illness—rampant. A commanding officer with a high sense of duty as he conceived it, which meant Spartan ideals that might have been less costly to human life if a little more time had been taken for acclimatization. "You are civilians! I will make you into soldiers!" And Lieutenant-Colonel Stawell, who stands up, looks over his glasses, and says in his staccato voice: "But you are not making them into soldiers, you are killing them!" That was courage.

These are the memories, the pictures etched on my mind, and on the minds of all of you who are old enough to have known him. Are they too intimate? I could have made them much more so, but that would be too poignant. To me they are the man in all his humanity!

Acknowledgement.

Most of the illustrations used in this lecture have appeared previously in *The British Journal of Surgery*, and I am indebted to the publishers of that journal for permission to reproduce them.

Reference.

(1) T. P. Dunhill: "Diaphragmatic Hernia", *The British Journal of Surgery*, Volume XXII, 1935, page 475.

Legends to Special Illustrations.

FIGURE I.—Stomach (1) and portion of colon (2) in the thorax.

FIGURE II.—The whole stomach is in the thorax, the greater curvature uppermost. 1: Pyloric portion of stomach; 2: Breast shadow.

FIGURE III.—The fundus of the stomach (1) is seen almost reaching the sterno-clavicular articulation. 2: Colon.

FIGURE VI.—Lateral view. 1: Contents of sac. 2: Œsophagus. 3: Level of diaphragm.

FIGURE VII.—1: Terminal coil of ileum. 2: Appendix and colon. 3: Neck of hernial sac at site of the defect.

FIGURE X.—Skiagram showing that the caecum has returned to its normal position.

FIGURE XII.—The œsophagus is seen entering the cardiac end of the stomach, which is directed cranially. The main part of the stomach is situated in the thorax and is filled with gas. This has displaced the heart to the right (shadow seen on the left of the film).

FIGURE XIII.—The œsophagus enters the stomach in the abdomen; the stomach at once enters the thorax. A narrow line of barium indicates its exit from the thorax, the pyloric portion of the stomach being in the abdomen. The aperture in the diaphragm is shown in Figure XI.

FIGURE XV.—The same patient as in Figures I and II after operation. 1: Fundus of stomach. 2: Prepyloric segment. 3: Duodenal cap.

FIGURE XVI.—Stomach herniated through the right dome of the diaphragm.

FIGURE XVII.—Same patient as in Figure XVI after operation.

FIGURE XX.—Œsophagus of normal length. The stomach is in the posterior mediastinum.

FIGURE XXI.—The stomach occupying the sac in the posterior mediastinum. Same patient as in Figure XX.

FIGURE XXIII.—Para-œsophageal hernia. Œsophagus normal length. The thoracic loculus of the stomach pushes up and deflects the œsophagus.

FIGURE XXIV.—Stomach with greater curvature uppermost; pylorus fixed above diaphragm; barium leaving pylorus in a thin trickle.

FIGURE XXVI.—1: Œsophagus. 2: Cardia. 3: Thoracic loculus. 4: Dome of left diaphragm.

FIGURE XXVII.—Dilated œsophagus; cardia in the thorax; portion of stomach in the thorax. Both domes of the diaphragm are seen.

FIGURE XXVIII.—Showing dilated œsophagus, a narrow stricture at the cardia with an ulcer above the narrowing, the cardiac portion of the stomach in the thorax.

FIGURE XXIX.—The same patient as in Figure XXVIII one year later. Ulceration deeper.

FREQUENCY OF MICTURITION IN THE FEMALE.[1]

By W. JOHN CLOSE, M.B., M.S., F.R.C.S. (Edinburgh), F.R.A.C.S.,
Adelaide.

I HAVE been induced to choose as my subject this evening a common symptom complex which affects so many women that it is an everyday complaint in the surgery of the general practitioner. The paucity of information in most urological and gynæcological reference books on minor female urinary ailments indicates the need for a clinical classification, and one is then thrown back mainly on personal experience to distinguish the various pathological conditions, their causes and treatment.

Admittedly, from time to time surgeons displaying a particular interest in the lower part of the abdomen have discussed these troublesome ailments, and occasional articles have appeared in periodicals; but these are not readily accessible to the busy practitioner, whose library naturally must be of an encyclopædic nature.

I shall, therefore, in this paper endeavour to censor the dogmatisms of the respective urological and gynæcological enthusiasts and summarize a variety of pathological conditions from a purely clinical aspect. Though humbly conscious at the same time of my deficiencies in the branch of microscopic pathology, I can at least assure you that I have made a serious attempt to reconcile my clinical findings with the pathological researches of others.

I realize also that I am encroaching on the front line trenches of my gynæcological allies, but it is

[1] Read at a meeting of the South Australian Branch of the British Medical Association on April 30, 1936.

at this point that we make contact in a common offensive to relieve female distress, and the effectiveness of such a liaison is assured only by presupposing that each possesses more than a smattering of the language of the other.

So usual is frequency with more or less dysuria or incontinence in the female sex that the sufferer has learned to regard her disability as her natural heritage and frequently fails to mention it to the doctor, except in reply to leading questions, or until her sleep, or perhaps her husband's, is seriously interfered with. The truth of this may be realized if out-patients are interrogated as a routine about their bladder functions, even though the main complaint may be, say, a Colles's fracture. The reply will often too truthfully be: "Oh, passable", which is all they have learned to expect of their refractory sphincters.

It is in the lower part of the urinary tract that the lesions responsible for the ailments peculiar to females occur, that is, that part which developmentally belongs to the Wolffian duct—the trigone, urethra and their adnexal glands. The female has no priority claim to any pathogenic state arising above this area, so that these latter, however interesting some of them may be, should not come into tonight's discussion.

Anatomy of the Female Trigono-Urethral Tract.

The urethra itself, five centimetres (one and a half inches) long, corresponds to the prostatic and membranous urethra of the male. It is surrounded by striated muscle fibres throughout practically its whole length, the *sphincter urethræ* or external sphincter, whilst a longitudinal layer of non-striped muscle lies between it and the submucosa; the longitudinal muscle spreads out fanwise in a backward direction through the trigone, as the trigonal muscle.

The internal sphincter, compared with the external, is an ineffectual loop surrounding the internal meatus; it is probably induced to open more by the pull of the trigonal muscle than by its own relaxation. Almost, if not more, effective as a sphincter is the normal acute angle between urethra and anterior vesical wall. Figure I is an adaptation of Young's diagram[10] of the male sphincter.

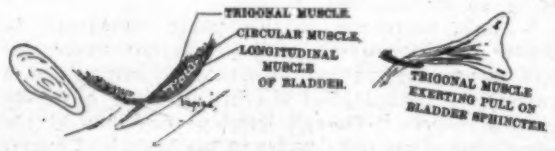

FIGURE I.
Diagram of bladder musculature in region of meatus. (Adapted from Young.)

The muscular mass comprised of the *compressor urethræ* and *transversus perinei profundus* is, for the sake of complicating description, referred to by Bonney as the "peri-urethral muscle wedge",[1] whereas it has long been known to anatomists as the urogenital diaphragm (Figure II).

Superior to this diaphragm is the pubo-coccygeus muscle, which Bonney appears to have ignored, covered inferiorly and superiorly by its epimyseal fascia, the superior layer assuming aponeurotic strength for the support of the genito-urinary organs. Where the two layers fuse at the rounded medial border of the pubo-coccygeus, the aponeurosis becomes cord-like in its strength and is usually the anterior end of the "white line of the pelvic fascia". This cord-like structure is the middle pubo-vesical ligament, and the aponeurotic sheet

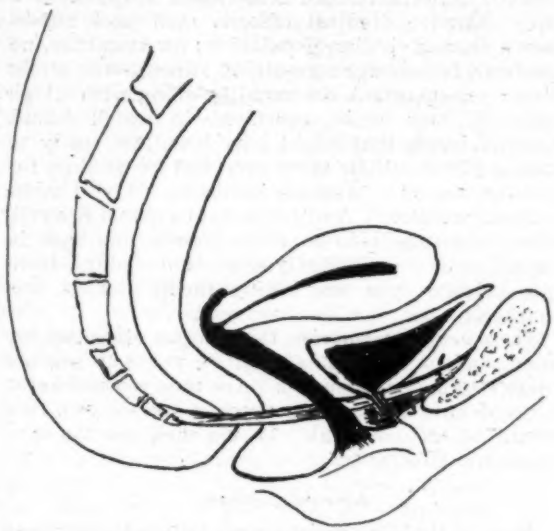

FIGURE II.
Diagram of supports of bladder and urethra. 1: Pubo-vesical or pubo-cervical ligament—Bonney's pubo-cervical muscular fascia. 2: Pubo-coccygeus muscle. 3: Urogenital diaphragm (*sphincter urethræ* and *transversus perinei profundus*)—Bonney's peri-urethral muscle wedge.

extending laterally towards the pubic ramus is the lateral pubo-vesical ligament of anatomists (in the old nomenclature "the lateral ligament of the bladder").

Bonney again ignores anatomical usage by coining the phrase "pubo-cervical muscular fascia" for this aponeurotic structure, and gynæcologists frequently refer to it, legitimately it would seem, as the pubo-cervical ligament. It may be granted that it is frequently just as muscular as it is fibrous, but the muscle fibres are those of the powerful pubo-coccygeus, and not involuntary muscle, as the above quoted authority states.

The dissection here shown (Figure III) indicates the support given to the bladder base and cervix by the attachments of the pubo-coccygeus and its aponeurosis; and it will be seen how the vesico-vaginal septum lies practically in the same plane as the pubo-vesical ligaments, although not morphologically continuous with them.

The epithelial lining of the tract under discussion commences at the trigone and posterior urethra as a

definite transitional type, gradually changing to a stratified squamous epithelium at the external meatus. Minute submucous glands are scattered through its length, homologous with Littré's follicles in the male, but the lacunæ of Morgagni found in the male are, in my experience, wanting in the female. As these occur in the roof of the penile portion of the male urethra, which is not represented in the female organ, it is not surprising that the lacunæ are absent too. However, certain crypts are sometimes to be found in the floor of the urethra, occasionally sufficiently large to form a diverticulum. Such diverticula, I suggest, may be caused by supernumerary ureteric buds growing out from the Wolffian duct; in fact a ureter itself is occasionally found implanted in the urethra.

Of most interest are the deeper glands situated below the longitudinal muscle layer. These are complex acinous and tubular glands commencing in the trigone and increasing in size and extent the closer they approximate to the posterior urethra, ramifying in the crossing muscle bundles, one or two fairly constantly perforating the urethro-vaginal septum. Their contents are homogeneous, resembling amylaceous corpuscles of the male prostate, even to the formation of concretions. This collection of glands is frequently referred to as the "female prostate".[1]

At the external meatus the two interesting Skene's glands are situated one on either side of the floor; the orifices of these glands become apparent only in the more or less prolapsed urethræ of parous women. Their long ducts pass backwards, parallel with the urethra, for one centimetre or more. These are the homologues of Cowper's glands.

Other para-urethral follicles may be seen in the vestibule near the urethra; these, I suggest, may be the homologues of the lacunæ of Morgagni.

The calibre of the urethra is about 30 to 35 Charrière (30 Charrière being equal to a diameter of one centimetre).

In the following description reference to the urethra will be understood to include the trigone as well.

Clinical Classification.

The following is a clinical classification of pathological conditions affecting the female urethra and trigone.

1. Urethritis.
   - *A.* Acute generalized infections due to:
     - (*a*) The gonococcus.
     - (*b*) A Gram-negative extracellular diplococcus.
     - (*c*) The *Bacillus coli*.
     - (*d*) The enterococcus.

     (May be found in pure culture.)
     - (*e*) Mixed organisms, such as staphylococci, diphtheroids and trichomonads, together with the above-mentioned organisms.
   - *B.* Chronic glandular inflammations of the deep "prostatic" glands, submucous follicles or Skene's glands. These may be:
     - (*a*) Infective, due to organisms mentioned.
     - (*b*) Non-infective conditions of the so-called "prostatic" glands.
     - (*c*) Passive venous congestion.
2. Stricture of the urethra accompanied by chronic infection as cause or effect.
3. Caruncule (these should be placed under *1B* as Skenitis).
4. *Trigonitis pseudo-membranosa*, or *trigonitis exfoliativa* (should be placed under *1B* (*c*).
5. Senile atrophic urethritis.
6. Diverticula.
7. Defects in the supports of the lower urinary tract.
8. Neuroses.

Urethritis.

Acute Generalized Infections.

Gonorrhœa, being no respecter of sex, need not be considered separately, but acute infections, apart from the Neisserian type, may, in my experience, be caused by the first three organisms mentioned and closely simulate gonorrhœa.

The *Bacillus coli* and enterococcus probably infect the urethra directly from the anus, and no doubt an ascending cystitis and pyelitis frequently occur from such a primary focus, accounting for the greater frequency of pyelitis in the female.

An interesting type is that due to the unclassified Gram-negative diplococcus which is not intracellular. It may produce a picture of gonorrhœa with its urethritis, cervicitis, bartholinitis and ascending metritis, though I do not know whether salpingitis ever occurs in this group.

I have been told that it is a very infectious condition in female children and, according to experience at the Children's Hospital, is more difficult to eradicate than the infection caused by the gonococcus; in the adult female, on the other hand, I have not so far found it intractable, though a stubborn trichomonal vaginitis may persist as a secondary infection.

With regard to the *Trichomonas vaginalis*, it has been my impression until recently that this organism in small numbers is a normal inhabitant of the vagina and that it becomes pathological in the female genito-urinary system only as a secondary infection. But Mr. S. F. Tee, of the Adelaide Hospital laboratory, who has recently done a great deal of research into trichomonal infections in association with Dr. B. H. Swift, does not altogether agree with this assumption. We may look forward with interest to the publication of their findings in the near future.

Gladys M. Sandes[7] described an intractable condition that was a source of continued frequency of micturition in young women, especially dancers and gymnasts. In one year she treated five games mistresses of girls' schools. Films showed masses of epithelial but no pus cells, and on culture diphtheroids were grown. Her treatment was by alkalis and glycerine instillations. The coincidence of these cases, rather than the organism, which most bacteriologists, I believe, would discount, interests me enough to make me mention them here.

[1] At this stage Dr. Close showed on the screen microscopic sections taken from Renner.[6]

Chronic Urethritis.

Chronic urethritic conditions interest me probably more than any other, since they occur in the greater number of my private female patients.

I have frequently seen patients who have suffered for many years from intense frequency of micturition and pain, with recurrent exacerbations of severity, when the diagnosis has continued to be pyelitis or cystitis. The two last-mentioned terms seem to be the garbage cans of indefinite urological states. It may be that at some time or other in the early history pyelitis has been present, again as an ascending infection, but when examined by myself the condition is purely urethral. The diagnosis is made by a process of exclusion and by the positive urethral findings of the urethroscope and cystoscope.

Such findings, I admit, do not always differentiate the condition from a chronic urethritis due to (*a*), (*b*) or (*c*) above, but the treatment is the same whether the cause is infective or not. There may be:

1. Most commonly a stunted polypoid condition of the internal meatus and bullous œdema of the distal part of the trigone. Various degrees of this polypoid condition may occur, from sessile masses containing well-defined vascular loops, and presenting a saw-edge effect of the meatus, up to frankly pedunculated projections.[4]

2. A mild granular condition of the urethral mucosa due to infection of the submucous follicles, with or without contracture of the lumen. Sometimes leucoplakia or small cysts are also seen.

3. An absence of urethroscopic signs, or signs so slight that they may be ignored. In these cases, on account of the symptoms and absence of signs elsewhere, I have been in the habit of giving the patients a course of routine treatment, and when relief occurs have been persuaded to include them in this category. This condition may be the result of some perimeatal scar tissue or venous congestion irritating the nerve supply of the muscles.

Urethral smears are not always obtainable, but in attempting the milking of the urethra one will usually find it more tender than normal; this applies also to the passage of an instrument, and the Kollmann's dilator may not readily expand to the normal 30 Charrière.

In the absence of smears I have often had recourse to a simple expedient to collect urethral secretions. The patient's vagina is first well douched, special attention being paid to the vestibule round the urethra. After drying, a tampon is placed in the vagina, the patient then being asked to pass her urine into two glasses, the amount in the first being a small quantity, about half to one ounce. This, when centrifuged, will frequently show pus cells, whereas the later urine may be free or may contain a relatively small number.

The causes of a chronic infection of the urethra may be so distant as to be uncertain, but so often according to the history it has commenced after childbirth. It may be that trauma had something to do with it, or catheterization during the puerperium.

Gynæcological operations have usually been repeatedly performed, particularly anterior colporrhaphy, perineal repairs and ventro-fixation, without success. I shall have more to say later about indiscriminate operations for cystocele.

Accompanying chronic urethral inflammation one frequently does find infections of the genital tract, such as some cervical erosion or a frank cervicitis, or leucorrhœa due to other causes.

Naturally, then, treatment must commence with that of the accompanying condition or recurrences are likely to occur; but it must not be expected, as I have seen stated, that this treatment alone will clear up the urethral condition.

Here the time seems ripe to draw attention to the habit of some surgeons of operating on a gonorrhœal salpingitis without paying attention to or providing for later treatment of the urethra and cervix. The external genital infections are far more important from the viewpoint of either the patient or her associates, whereas salpingitis becomes spontaneously sterile, even if untreated surgically.

The treatment of the urethra itself is usually much simpler than that of chronic urinary infections elsewhere. It consists in the use of the dilator, a straight Kollmann's for preference, and the instillation of silver nitrate solution of strengths from 0·5% to 5·0% or, in intractable cases, to 10·0%. One may even employ this strength on a dressed probe, actually massaging the urethral mucosa therewith; but I have seldom found this necessary.

Hegar's dilators are often efficient substitutes for Kollmann's dilator, working up to a corresponding size, but I have had a number of cases in which these dilators have failed and the Kollmann's has proved successful. The reason may be that an expanding dilator produces less trauma, acting rather as a masseur of the urethra. In fact the main plea in this paper is for the Kollmann dilator where treatment is discussed, and you will hear it recurring in this production like the theme song of a modern screen drama.

Usually 30 Charrière is sufficient, but sometimes 35 has been reached. The normal urethra I find is seldom larger than this, whatever some authorities may say as to its dilatability. One surgeon recently stated that he dilated to 45; but this is almost as large as the proximal knuckle of my little finger, and I think it is courting disaster.

Some years before I took a special interest in urology I dilated a urethra for a very distressing condition such as I have described, finishing up with my little finger. I had seen it done before. I cured the patient's dysuria, but not her frequency, for she failed to hold a drop of urine in her bladder after that. She was well pleased, however, at the time; but Dr. W. A. Verco later spent a lot of energy trying to remedy by surgery the effects of my enthusiasm, without any immediate result. He may be pleased, however, to know that his efforts were eventually crowned with success after the lapse of some years.

Instillations are given with a very blunt-nosed rubber ball type of syringe, the solution being squirted through the urethra from the external meatus after dilatation.

ILLUSTRATIONS TO THE ARTICLE BY SIR THOMAS DUNHILL.

Figure I.

Figure II.

Figure III.

Figure VI.

Figure VII.

Figure X.

Figure XII.

Figure XIII.

Figure XV.

ILLUSTRATIONS TO THE ARTICLE BY SIR THOMAS DUNHILL.

Figure XVI.

Figure XVII.

Figure XX.

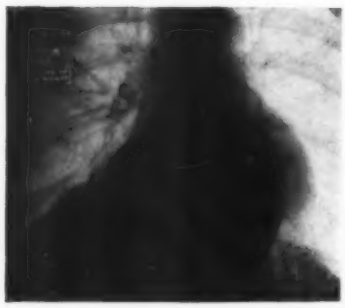

Figure XXI.

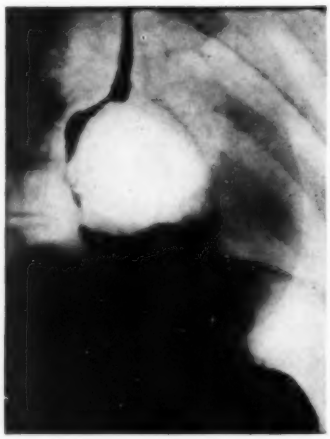

Figure XXIII.

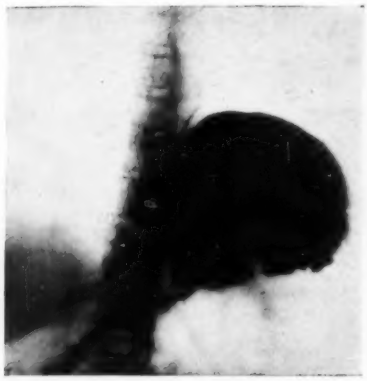

Figure XXIV.

Figure XXVI.

Figure XXVII.

Figure XXVIII.

Figure XXIX.

Skenitis.—Skenitis is a common accompaniment of the deeper lesions, but frequently an inflammation of Skene's ducts is the only demonstrable lesion. In the most pronounced cases a definite redness and prolapse of the lower half of the meatus are seen, or a granular vascular appearance up to a definite caruncle presents in the interval between the ducts. I shall have more to say about this when considering caruncles, which I have given a separate heading only because of custom, but which I believe should really be discussed with skenitis.

Whether the more extreme degrees of neighbouring reaction are seen or not, the meatus is as a rule extremely tender to touch, and this may absolutely prohibit sexual intercourse.

The duct openings may or may not be seen, depending on the amount of swelling which tends to hide them. If they are visible, pus may be expressed from the orifices, though sometimes only after repeated attempts; and even if they are not visible the pus may be seen to exude from the lateral aspects of the swelling. Such attempts at expression may sometimes be undertaken only during anæsthesia.

Suppurative skenitis is not an uncommon condition, although Kidd[5] stated that he found it only twice in 650 cases, one of which was gonococcal and the other not. On the other hand, most of the gonorrhœal patients whom I see have some Skene's infection, which usually outlasts the rest of the urethral infection. Dr. H. M. Fisher tells me that his experience as to the frequency of skenitis in the night clinic is similar.

Non-gonococcal skenitis is also frequently seen, the organisms being usually mixed.

In one case referred to me by Dr. Fisher *Bacillus coli* was present in pure culture; one duct only was distended into a cystic dilatation within the urethra, and from this a drachm of pus could be expressed. The cyst was destroyed and the condition was cured by diathermic coagulation.

Treatment of chronic skenitis depends on destruction of the ducts. This may sometimes be done by the fused silver nitrate probe, though rarely in my experience. Slitting the glands is possible only if the degree of swelling allows it to be done; but if so, a bistoury such as that used for the lachrymal duct is a useful instrument, followed by cauterization of the tract.

When swelling, pain and a granulomatous condition have commenced, I have found it necessary to resort to excision. The operation will be described in the discussion of caruncles.

Non-Infective Conditions of the So-Called Female Prostate.

It would appear that the deep tubulo-acinous glands that have been called the female prostate undergo many of the changes to which the male prostate is subject. These include: infections; dilatation of the acinæ into small cysts; calcification of *corpora amylacea* to form small concretions which, projecting on the surface of the meatus, produce a great deal of irritation; and fibrosis.

Some of the difficulties associated with senile irritability in which atrophy and contracture are noticeable may be due to such fibrosis round these glands. Adenomatous hypertrophy such as one finds in elderly males is, however, not described; the reason possibly is that some hormonal stimulus is wanting.

The largest calculus of the kind described which has come under my notice was the size and shape of a peach kernel. It was completely surrounded by mucosa, which provided it with a stalk, so that it projected almost to the external meatus. The patient's complaint was almost complete retention of urine.

Treatment is again mainly by the Kollmann's dilator. The extrusion of small calcareous bodies can also be encouraged by massaging the urethra through a urethroscope cannula with a dressed probe. Large calculi, of course, must be excised if within reach, or, if too deeply situated, released by the cautery.

Passive Venous Congestion.

Frequency of micturition is a common symptom in the physiological venous turgescence of pregnancy and in pathological states which interfere with the return of blood from the pelvic veins.

Some glandular catarrh would naturally be expected at the neck of the bladder, so that if relief does not follow treatment of the causative condition, the Kollmann's dilator again comes into its own. A persistent varix is sometimes seen on the trigone and may have to be touched with a diathermy electrode.

Stricture of the Urethra.

Stricture of the urethra is said by some observers to be as frequent as stricture in the male. If so, it does not become such a formidable obstruction as a rule, for I have seldom seen the female urethra contracted to less than 18 Charrière. There is usually present some granular urethritis, which is the causative condition, and infection of the deeper glands behind it, which is the resulting state.

The symptoms are thus more irritative as a rule than obstructive. The greatest contracture is likely to occur following gonorrhœa. The treatment is the same as that for chronic urethritis, whether gonorrhœal in origin or not, namely, use of the Kollmann's dilator and silver nitrate instillations.

Caruncular.

A caruncle of the urethra is a vascular, exceedingly tender, sessile or pedunculated granuloma situated in the lower half of the external meatus. Microscopically, extreme thickening of the stratified epithelium, resembling an epithelioma with its downgrowths and papillæ, is found. Dilated vessels, plasma cell and polynuclear cell infiltration of the epithelial columns and round cell infiltration of the stroma complete the picture. The polynuclear cell infiltration and the packing of the small vessels

with polymorphonuclear cells suggest an infective origin.[1]

Ewing[3] writes:

A common error in diagnosis of urethral caruncule in females results from the deep normal invagination of the epithelial lining of this structure. On section these invaginations may appear as displaced islands of a typical epithelium strongly resembling epitheliomata.

Charles Olcott submitted several of these growths to Ewing for diagnosis when sections had been proclaimed malignant by other pathologists; in each case Ewing affirmed that they were innocent.

It seems probable that many of the so-called carcinomata of the external meatus are therefore of this pseudo-epitheliomatous type.

The cause has been variously guessed at, but many authorities suggest an infective origin. My own observations of the constant association of Skene's duct infection with this condition, and the different degrees of reaction in the neighbouring meatus which I have described above, lead me to regard the caruncle as a polypus arising as a result of chronic suppuration in these ducts.

On searching the literature for some corroboration I find that Olcott holds a similar view. In a series of twenty-three cases examined microscopically he found glandular elements in seventeen.

Treatment.—I commenced treating these growths by diathermic coagulation, as is usually recommended, but I found, after shrinkage of the growth, that the Skene's ducts remained grossly infected, that the meatus was still excessively tender, and that the growth soon began to reappear.

I now waste no time over cauterization methods, but dissect out the lower half of the meatus, including caruncle and glands, taking care that the ducts are not cut across, but removed completely. The cut urethral floor is then brought down to the incision in the vestibule and united in a transverse line by interrupted catgut sutures. The same operation is performed for chronic skenitis when it is not possible to destroy the ducts by cauterization or when the reaction is such as to suggest a commencing carunculosis.

I have not found stricture or prolapse follow this procedure, with the exception of one case, in which prolapse occurred after the dissection of the whole circumference of the meatus.

Trigonitis Pseudo-Membranosa or Trigonitis Exfoliativa.

Trigonitis pseudo-membranosa, seen only on the trigone of women, is a pearly or cloud-like raised appearance of the epithelium of the trigone. It has a creeping, apparently semi-detached, irregular edge, which appears to be growing over the trigone radially from the meatus; it never, I believe, spreads beyond the trigone. Sometimes the surface under its semi-detached edge is seen to be slightly reddened or excoriated, like the base of a patch of psoriasis. Like psoriasis, too, it is usually chronic, but may occasionally be acute.

[1] At this stage Dr. Close showed on the screen microscopic sections taken from Olcott.[2]

The symptoms are more or less frequency of micturition and pain.

The cause has puzzled urologists and that most frequently given is an irritative bacilluria. In most cases, however, there is no abnormality in the urine beyond an excess of bladder epithelial cells.

I think it is fairly certain that this condition never occurs except as a result of present or past pelvic congestion, which is my reason for wishing to place it under heading 1. Therefore, when it is seen, a search should be made for uterine displacement, fibroids, diseased adnexæ or chronic cervicitis. These conditions may have been cured, but still the bladder appearances persist, and even possibly the symptoms.

So often one sees some *trigonitis exfoliativa* without many symptoms that I am persuaded that the symptoms depend on congestion of the meatal area and posterior part of the urethra rather than on the membranous condition itself; and the dilator has again been effective in clearing them up.

It seems to me that one can draw an analogy between this condition and the scaly dermatoses found on varicose legs.

Over two years ago I saw an especially acute condition of this nature. The patient, a woman of twenty-six years, was sent to me by Dr. C. Yeatman, complaining of gross hæmaturia, frequency of micturition and severe dysuria of three months' duration. The peeling, wavy outline of the pearl-like layer extended to the ureteric orifices on each side, was stippled here and there by ecchymotic patches, and blood oozed from the under-surface of the detached edge. *Per vaginam* a mass was felt in the left fornix, and on questioning the patient it appeared that the hæmaturia had a distinct relationship to her menstrual periods. The patient was referred back to Dr. Yeatman after some local cauterization, and he removed a large endometrioma of one ovary. Another nodule, he said, was excised from the vaginal vault. There has been no further trouble, I understand.

I am aware that endometriomatous nodules may appear on the mucosal surface of the bladder itself, but I have not seen them, and this case was decidedly not one of that nature. One would expect such nodules to lie deep to the trigone or bladder base, as they would no doubt be due to inclusion of endometriomatous cells in the embryonic cul-de-sac, which later fuses to form the vesico-vaginal septum.

Senile Atrophic Urethritis.

There is little to say about senile atrophic urethritis, except that caution is necessary in the use of the dilator; if it is used with discrimination it will often give at least temporary relief. Achlorhydria, I believe, may be associated with a similar atrophic condition, causing very irritative symptoms. The treatment is naturally of the associated condition.

Defects in the Supports of the Lower Urinary Organs.

The patient often quotes her doctor, correctly or not, as saying that her womb is pressing on her

bladder. Knowing how accustomed is the bladder to providing a resting place for weary and heavily laden organs, we can discount this as a cause of frequency of micturition.

Uterine displacements with accompanying venous engorgement have been dealt with above.

Cystocele I do not consider is a cause of urinary discomfort as often as supposed, and I should not recommend colporrhaphy for the cure of frequency of micturition or recurrent pyelitis unless residual urine and a basal cystitis are present, as shown by the cystoscope.

Stress incontinence is a condition to which attention was first drawn by Berkeley and Bonney; according to the latter, its pathogenesis is quite different from that of cystocele. It is due to the dropping of the vaginal attachment from the sub-pubic angle, by the relaxation of the pubo-vesical ligaments (or, as Bonney calls them, the pubo-cervical muscular fascia). The diagrams (Figure III) are taken from his description.[2]

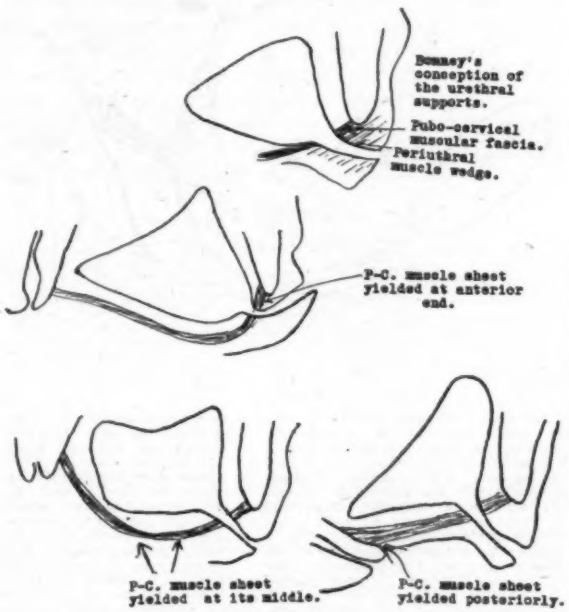
Bonney's conception of the urethral supports.
Pubo-cervical muscular fascia.
Periurethral muscle wedge.
P-C. muscle sheet yielded at anterior end.
P-C. muscle sheet yielded at its middle.
P-C. muscle sheet yielded posteriorly.

FIGURE III.

Stress incontinence is characterized clinically by the ejection of urine with expiratory effort, and in the worst cases with any movement in the upright position. If one examines the patient while she is upright, the lower end of the vagina protrudes on expiratory effort and the urethra tends to wheel round the suprapubic angle, so that the external meatus looks upwards and forwards, and a spurt of urine accompanies the excursion. In the normal woman the anterior vaginal wall "tautens upwards" owing to the automatic contraction of the pubo-cervical muscular fascia. Of course Bonney means the pubo-coccygeus muscle which controls the tension of the aponeurotic structures and which, incidentally, I believe all gynæcologists make use of in their repair work, even though they call it fascia or ligament.

Such a relaxation as that described no doubt exists, but Bonney's anatomical confusion is not clarified by the operation which he describes, of "tautening the urethral sphincter". Here the urethra is denuded of its vaginal covering and the muscles of the urogenital diaphragm (his peri-urethral muscle wedge) are pulled across Lembert-wise. This, he says, in some way tightens up a structure which lies superiorly and which runs in an exactly opposite direction. One can believe that the success of his operation is due purely to the effect on the urogenital diaphragm and that it is unnecessary to bring deeper structures into the discussion at all. Ventrofixation of either the uterus or bladder is mentioned as an alternative procedure. Reference to my dissection of the pubo-coccygeus again (Figure IV) will show the possibility of tautening the fascia in question by an anterior colporrhaphy and at the same time pulling the muscle sling together, just as one does posteriorly in the cure of a rectocele.

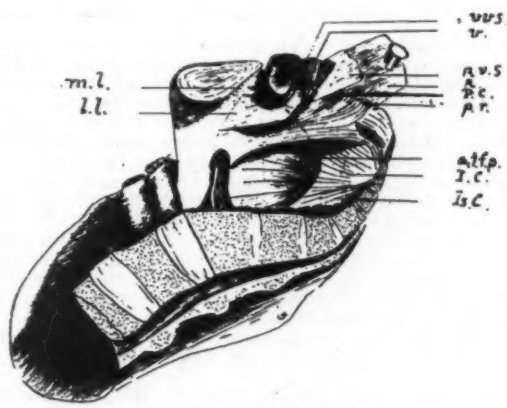

FIGURE IV.

Dissection of female *levator ani* muscle, primarily undertaken to show its attachments to all the pelvic viscera. The rectum is everted and pinned down to show muscle attachment; the hemisected vagina and bladder base are also slightly everted. The aponeurosis has been cleaned off the muscle, except anteriorly, where the striated fibres of this muscle blend. *l.l.*: lateral ligament of bladder (O.T.), lateral pubo-vesical ligament (B.N.A.), pubo-cervical ligament (gynæcological), pubo-cervical muscular fascia (Bonney); *m.l.*: middle pubo vesical ligament (B.N.A.); *v.v.s.*: vesico-vaginal septum; *r.v.s.*: recto-vaginal septum; *a.t.f.p.*: *arcus tendineus fasciæ pelvinæ*; *v.*: vagina; *r.*: rectum; *P.C.*: pubo-coccygeus; *p.r.*: pubo-rectalis, artificial subdivision of pubo-coccygeus; *I.C.*: ilio-coccygeus; *Is.C.*: ischio-coccygeus or coccygeus muscle. Note: The medial pubo-vesical ligament (B.N.A.) is a small mass of non-aponeurotic endopelvic fascia occupying the mid-line between *symphysis pubis* and bladder, serving only as a sheath for the pudendal venous plexus (Santorini).

Diverticula.

I have never seen a diverticulum of the urethra in the female, though I am constantly on the look-out for them. An infected cavity, with or without calculi, results in urinary irritability. Treatment, of course, is by excision.

Neuroses.

Bonney writes:[1]

Frequency due to functional disturbance of the vesical nerves never wakes her up; once she is asleep frequency ceases. Frequency which wakes a patient up has in all probability a gross physical basis.

The aphorism is worth remembering.

We may have under this heading two types: (*a*) Functional polyuria. (*b*) Hypersensitiveness of some part of the nervous mechanism, either situated at the micturition centre of the spinal cord or in the hypogastric nerves. Such a condition is termed by Bonney "urge incontinence", to distinguish it from the above-mentioned "stress incontinence". Any excitement or emotion tends to increase the frequency. I assure you that a young woman so afflicted makes a poor examination candidate. The treatment I suggest, after ruling out all possible organic causes, is either to stimulate the parasympathetic nerves, so as to improve the balance between sphincter and detrusor, or to dull the bladder's sensory nerves, the afferent part of the reflex arc.

The first effect I have found can sometimes be obtained by ephedrine and is worth trying.

A case illustrating such a condition was that of a woman aged forty-seven years, who from childhood had suffered from urge incontinence. During the day the intervals would be twenty minutes, though while she was at rest they might be two hours; at night she seldom rose at all (which fits admirably Bonney's epigrammatic description of the neurotic patient). She had also been subject to migrainous attacks since childhood. She had learned to put up with her frequency, but latterly had become keen on golf. Obviously her defect rather cramped her style, hence her belated search for relief. An exhaustive examination failed to reveal any organic cause, so ephedrine was administered in 0·03 gramme (half grain) doses daily. The effect was immediate. Twelve months later she reported that her urge to micturate had not interfered with her golf since then, and that even on the putting green she felt quite safe. In her own words the relief was complete.

For cases which do not react to this treatment I should advise the trial of spinal anæsthesia of the sacral area by means of heavy "Percaine" solution, with the object of damping sensory stimuli. Unfortunately I have not tried this method myself, but am just awaiting a suitable case. It was suggested to me by the occurrence of some slight difficulty of micturition in other patients who had been submitted to this type of anæsthesia for cystoscopic purposes, and by the relief which has occurred under similar conditions in certain types of severe strangury. After such an examination I have found more than once that the terrible pain of bladder carcinoma has been relieved considerably.

Surgical Attack on the Nerves of the Bladder.

Before drawing to a close I should like to digress somewhat and break a resolution made at the beginning, namely, of confining myself to essentially female ailments. The mention of carcinomata suggests the surgical relief of pain by resection of the presacral nerve.

The dissection on the table (Figure V) shows the sympathetic and parasympathetic innervation of the pelvis. From the aortic plexus the presacral nerve descends to the superior hypogastric or presacral ganglion, situated, as the name implies, on the anterior aspect of the body of the sacrum. Up to this point the whole of the sympathetic system lies embedded in a layer of fascia, shown in the dissection of another pelvis on the table, which, for the want of a name, I have called the presacral fascia. It is distinct from the *fascia propria* of the rectum, is easily and naturally separated from it in lifting the rectum away from the sacral hollow, as one does in the abdomino-perineal operation for resection of the rectum, and is obviously a vehicle or sheath for

FIGURE V.

Diagram of hypogastric ganglion receiving the sacral autonomics in the utero-sacral ligament. Sagittal section through sacrum to left of anterior sacral foramina and through body of pubis. P: pubis; B: bladder; R: rectum; C: cervix; S: sacrum; P.C.: portion of pubo-coccygeus muscle; 1: inferior hypogastric ganglion; 2: sacral supply from S 2 and 3; 3: autonomic supply to cervix, vagina and bladder base; 4: *nervus erigens*; 5: nerves to *levator ani*; 6: left inferior hypogastric nerve; 7: superior hypogastric or presacral ganglion; 8: pelvic colonic nerve; 9: superior gluteal nerve; 10: sacral plexus; 11: pudendal plexus. Structures 9, 10 and 11 are pinned back.

the nerves only. Between the presacral ganglion and the superior hæmorrhoidal periarticular plexus of nerves there is a connecting strand of fibres which has to be torn through when one attempts to separate the rectum and its *fascia propria* from the presacral layer.

This strand, by the way, is probably the parasympathetic nerve to the pelvic colon, ascending from the sacral pelvic nerves via the hypogastrics and inferior mesenteric plexus. This was recognized by Trumble[9] and by Telford and Stopford[8] to be the route taken by this particular nerve supply—Trumble's pelvic colonic nerve.

What I wish to point out mainly here, however, is the fact that the hypogastric nerves, after diverging on each side from the presacral ganglion,

pass into the utero-sacral ligaments, or, in the male, into genito-sacral folds. In fact these nerves, with their gangliform expansions, the inferior hypogastric ganglia, are responsible for these elevations, a fact which is not generally recognized even by gynæcologists.

Within the utero-sacral folds the sacral nerves supply their branches to the hypogastric ganglion, whence all the pelvic viscera, except the pelvic colon, receive their innervation direct. Now the bladder's sensory nerves are partly hypogastric (the fundus) and partly sacral (trigone, meatus and urethra).

The operation usually performed for the relief of bladder pain due to incurable conditions is resection of the presacral nerve and ganglion. In such treatment the sacral nerves are ignored, which may account for some partially satisfactory results.

I would suggest that the comparatively simple procedure of resecting portion of the utero-sacral ligaments would insure the division of both groups of sensory nerves. There seems to be no objection from the point of view of functional disturbance that cannot be made against the presacral operation—the pelvic colonic parasympathetics, for instance, would be divided in both cases. At the same time one might question the advisability of dividing these ligaments close to the cervix or more posteriorly, as recommended in descriptions of certain popular gynæcological operations.

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AN OPERATION FOR THENAR PARALYSIS.

By N. D. ROYLE, M.D., Ch.M., F.R.A.C.S.,
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THE loss of the intrinsic thenar muscles leads to a serious disability of the hand, namely, inability to oppose the thumb to the other fingers. The functions of the short flexor and the short abductor are relatively unimportant when compared with the function of the *musculus opponens pollicis*, but the functions of all these may be substituted by a transplantation which I have been practising for many years.

In looking round for a suitable tendon to transplant, it struck me that as the thumb had to be drawn to the medial side of the hand a muscle which normally flexes one of the medial fingers to oppose the thumb would be suitable. Therefore, I transplanted the short flexor of the ring finger.

Technique of Operation.

Step I.—To transplant the short flexor of the ring finger the skin is opened through the transverse fold at the base of that finger. By retracting the skin distally the insertion of the bifurcated tendon of the *flexor sublimis* may be reached. It is then cut through at each side (Figure I).

Step II.—Next, an incision is made through the deep fold proximal to the metacarpo-phalangeal joint of the thumb and the sheath of the *flexor longus pollicis* is opened (Figure I).

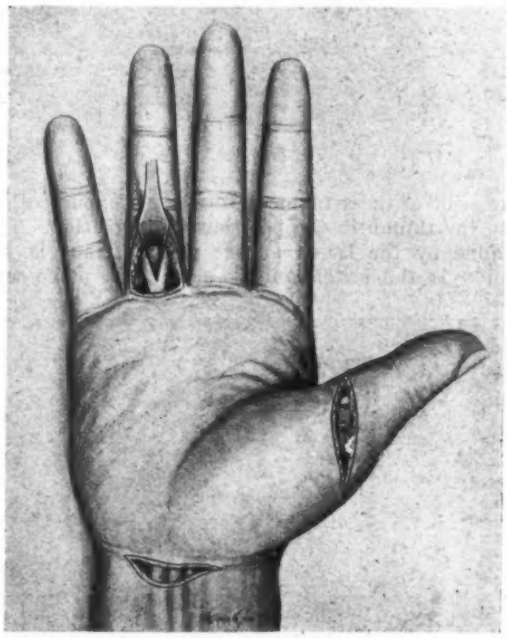

FIGURE I.

Step III.—An incision is made in one of the transverse folds of the palmar aspect of the wrist. The superficial flexor of the ring finger is then drawn out of this incision and passed up the sheath of the long flexor of the thumb and brought out of the sheath of this tendon (Figures II and III).

Step IV.—The skin is then dissected back over the attachment of the short flexor and the *opponens pollicis*. One part of the bifurcated tendon of the superficial flexor of the ring finger is stitched to the attachment of the short flexor, with the first phalanx in flexion, and the other part is stretched to the

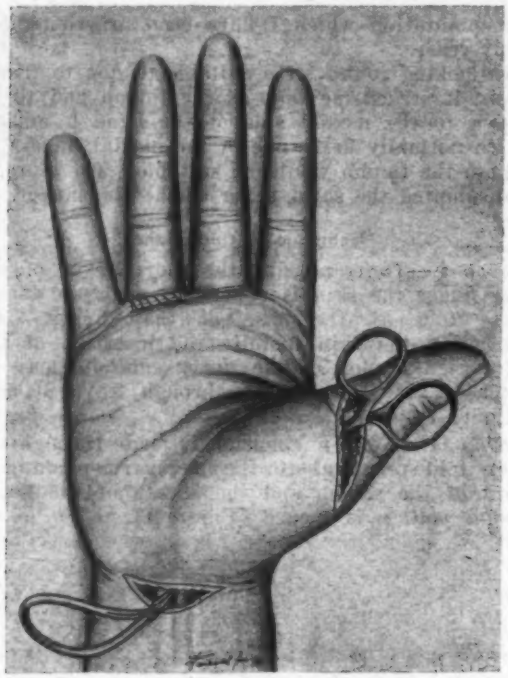

FIGURE II.

outer side of the attachment of the *opponens pollicis* with the thumb in the position of opposition. This is done by the living suture method, that is, the tendon is the stitch and is sewn into place with

FIGURE III.

the author's living suture needle. The tendon is actually threaded in the needle and sewn onto its new attachment. If the stitches are too short, the tendon of the *flexor sublimis* may be split so as to continue the bifurcation (Figure IV).

The living suture needle is like a Gallie's needle, but has a cutting triangular point. It is procurable from Messrs. Allen and Hanbury's and is made in four sizes: small, medium, large and extra large.

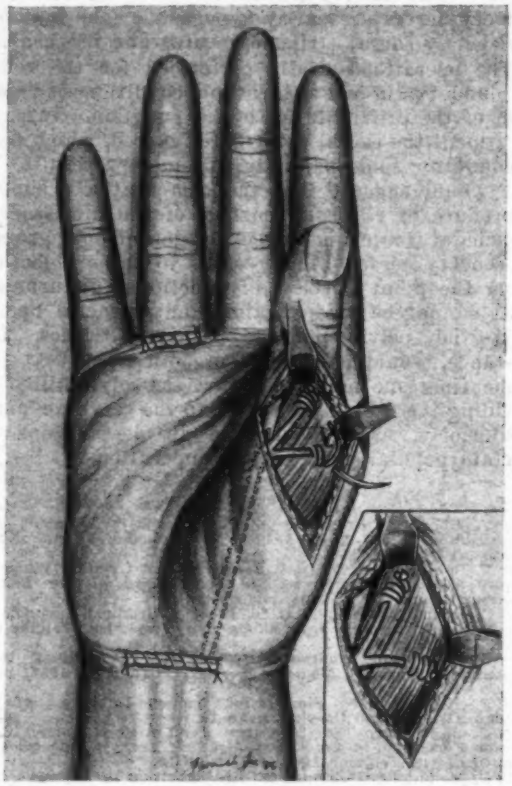

FIGURE IV.

After the wounds are closed and the dressings are applied the thumb is strapped in position of opposition and the flexion of the first phalanx and is kept in this position for ten days.

Reeducation.

Reeducation is easy, for when the patient attempts to flex his ring finger the thumb is drawn naturally into opposition to that finger.

The superficial flexor tendon of the little finger or middle finger may also be used for this operation, but the superficial flexor of the ring finger is less missed in the movement of the hand than that of either of the other fingers.

Acknowledgement.

The drawings in this paper are the work of Mr. David Farrell, of the Medical School, University of Sydney.

Reports of Cases.

EXTRADURAL ABSCESS SECONDARY TO A CARBUNCLE OF THE WRIST.

By M. E. CHINNER, M.B., B.S.,
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ON August 11, 1935, M.R.R. came to my consulting room with a small infection of the wrist, which was treated with hot foments. He reported again on August 13, when pus was present, and a small carbuncle was incised and treated with glycerine and magnesium sulphate. He was seen again on August 15, when the sloughs were separating, and he said that the wrist felt much better. He next reported on August 20 for general treatment and said casually that he had a slight backache in the lumbar region. He sent a message on August 25 that he had a very severe backache and was unable to visit my rooms; but there was no sign of any nervous lesions, although he was diffusely tender in the lumbar region and had extra pain on sitting up and bending his back; this condition was much the same the next day. I was unhappy regarding his condition and saw him early on the morning of August 27, when I found that during the night he had gradually lost the power of moving his right leg; his left leg was definitely weak, and he had sensory changes which pointed to trouble at about the eighth to the tenth dorsal segment.

I then had a consultation with Dr. C. T. de Crespigny and the patient was removed to hospital and a lumbar puncture was performed. The pathologist's report was that the fluid had a pale yellow tint. An excess of globulin was present. The protein was 880 milligrammes per 100 cubic centimetres of cerebro-spinal fluid (normal is 15 to 45 milligrammes). Examination of a film revealed many erythrocytes and some polymorphonuclear cells, probably rather more than could be accounted for by the blood. No microorganisms were detected, and later no growth was obtained on attempted culture. The findings were compatible with spinal compression.

An X ray examination of the vertebræ was carried out and the radiologist reported that no abnormality could be detected.

The white blood cell count was 37,000 per cubic millimetre.

By midday of August 27 the paralysis was rapidly increasing and the patient had a complete paraplegia.

Sir Henry Newland was consulted and was of the opinion that the paraplegia was due to compression by inflammatory exudate secondary to osteomyelitis of the spine. He considered that an immediate laminectomy was advisable, with the object of relieving the pressure on the spinal cord and of providing an exit for the pus. At 8 p.m. on August 27 Sir Henry Newland performed an extensive laminectomy involving the eighth, ninth, tenth, eleventh and twelfth dorsal vertebræ. Beyond some congestion of the peridural fat nothing of significance was found on removing the laminæ of the upper two vertebræ. When the laminæ of the tenth dorsal vertebra were removed, a small stream of thick, creamy, yellow pus welled slowly from the reddened peridural fat. On a small scale, the appearances were those of a perirenal carbuncle with its small areas of pus. (On culture the infecting organism was found to be *Staphylococcus aureus*.) The removal of the laminæ of the last two dorsal vertebræ revealed the same condition. The bodies of the vertebræ were drilled via the pedicles, but no pus was discovered. The long wound was then closed, provision being made for drainage. In the absence of radiographic and operative evidence of infection of the bone, Sir Henry Newland came to the conclusion that the cause was analogous to that of a perirenal carbuncle and that the peridural fat had become infected secondarily to the carbuncle on the wrist.

On August 28 the patient had a little movement in the left foot, but he had retention of urine and all the classical sensory disturbances associated with paraplegia. On August 29 he had a little more movement in the left foot; the white blood count had fallen to 18,000 cells per cubic millimetre.

The patient's general condition was still fairly desperate, with a rapid pulse, swinging temperature, and no improvement in the paralysis of the right leg; so Sir Henry Newland decided to remove the laminæ of the first and second lumbar vertebræ. This was done, but no further pus was discovered.

The first sign of returning power in the right leg occurred on September 1, and from that time onward the legs gradually improved; but the patient's bladder did not empty naturally until September 20; until this time his bladder was being catheterized twice daily.

From September 20 to October 2 the patient was allowed to sit in a chair and gradually began to walk a few steps with the aid of two sticks. He was discharged from hospital on October 2. His convalescence was extremely slow and he was treated on constitutional lines, his main symptoms during that time being backache and a sense of weakness, with a fair amount of spasticity in both legs, more so in the right. I had a large leather jacket made to support his back, and its use was attended by much improvement.

His improvement was gradual and he was able to return to work as a school teacher early in December, 1935.

I have seen him on many occasions since. His left leg is normal and he has just a slight spasticity of the right leg. He walks well and briskly without aid of any kind.

The above-mentioned condition is extremely interesting and must be very rare. Pathologically it is comparable with perirenal abscess or solitary hepatic abscess.

Acknowledgements.

In conclusion I wish to thank both Sir Henry Newland and Dr. C. T. de Crespigny for their very expert and skilled assistance in this case.

Reviews.

CARDIAC OUTPUT.

IN a slender volume on cardiac output and arterial hypertension Dr. S. A. Gladstone has published separately the results of some work carried out as Sutro Fellow in Cardio-Vascular Research at the Mount Sinai Hospital, New York.[1] The experimental work, which has appeared in current journals, includes a critical examination of the Marshall-Grollmann method for the determination of cardiac output, a description of the modifications suggested by this examination, and an account of the application of the new method to two normal subjects and to five showing various degrees of hypertension. An hypothesis postulating a humoral control of blood pressure is put forward in the last section.

The Marshall-Grollmann method depends upon the measurement of the amounts of acetylene and of oxygen which simultaneously disappear from a uniform gas mixture in the lungs. The amount of acetylene disappearing is a measure of the volume of blood necessary to carry it away. The amount of oxygen disappearing is that taken from this volume of blood by the tissues. A determination of the rate of oxygen consumption of the subject enables the rate of cardiac output required to furnish this amount of oxygen to be calculated. It is essential that the first part of the measurement be completed before any blood which has been exposed to the gas mixture has time to return to the lungs. A period of twenty-three seconds is required for this part of the measurement.

[1] "Cardiac Output and Arterial Hypertension", by S. A. Gladstone, M.D.; 1935. New York: Published by the author. Royal 8vo, pp. 56, with illustrations. Price: $1.00 net.

The author has shown that the deep breathing necessary for mixing the gases in the lungs nearly doubles the rate of the pulmonary circulation, that some blood begins to return to the lungs in about ten seconds, and that very considerable quantities have repassed the lungs before the lapse of twenty-three seconds. The author has shown further that the criterion used for determining when the mixing of the gases in the lung is complete has greatly exaggerated the difficulty of this process. Neglect of the effect on the total volume of variations of the apparent respiratory quotient (from above 1·5 to below 0·5) has been responsible for this.

A modification of the Marshall-Grollman technique has been employed in which the number of mixing respirations has been halved and in which the attainment of a homogeneous mixture is accelerated by preliminary inhalation of a gas mixture which at once brings the composition of the lung contents practically to that finally reached with the gas mixture used in the experiment. The whole procedure occupies eight seconds and the very serious errors due to recirculation of blood are eliminated.

The reproducible and apparently satisfactory results obtainable by the original method are shown to be possible by a balancing of the errors due to a slowing of the rate of absorption of acetylene on the return of previously exposed blood by the diminution of the absorption of oxygen from the blood owing to the accelerated circulation.

In view of the importance of a satisfactory method for the determination of cardiac output, it is rather a pity that more measurements on normal subjects were not published. Those published show excellent concordance and agree well with results by other methods. Comparisons between individuals would have been facilitated by giving cardiac indices (output in litres per minute per square metre of body surface), as these have been shown to vary within a much smaller range than total outputs.

The author's hypothesis on the humoral control of blood pressure assumes the production in the kidney of some unknown pressor substance, perhaps acting synergistically with adrenaline. In renal insufficiency of any origin this substance is assumed to accumulate in the blood by reabsorption until the rise of blood pressure increases the rate of urine formation to the point at which elimination of the substance again balances rate of formation. The hypothesis is not altogether novel, and while, as the author claims, it may do no violence to established facts, it leaves a great deal to the imagination. What, for example, controls the rate of formation of the pressor substance? Surely this is just as important as the mechanism controlling its rate of excretion.

PROGRESS IN DERMATOLOGY.

THE publishers of "The Recent Advances Series" are to be congratulated on their selection of Dr. W. N. Goldsmith as the author of the latest addition to the series. "Recent Advances in Dermatology" is a tribute to the erudition of the author.[1] In this book a staggering amount of the dermatological literature published during the last fifteen years or so has been absorbed and then presented to us in a most logical manner.

In the introduction the reader is advised to read straight through the book; anyone who does so will be well repaid. This is no ordinary text-book of dermatology and therefore cannot be recommended to the general practitioner in search of help in the diagnosis and treatment of a particular dermatosis. It can, however, and must be recommended to all dermatologists and to all who wish to keep abreast of modern dermatology.

At the conclusion of each chapter a bibliography is given. The earlier chapters deal with the finer and newer points of the physiology and pathology of the skin. Endocrine, nervous and mental influences are considered in a well-balanced chapter. A hint is given that the attribution of all kinds of skin disorders to psychological causes on flimsy evidence is most undesirable. Allergy, a subject often so mystifying to the average reader, is made clear, and the effects are exemplified in a discussion on eczema. The author has had the courage to give a definition of eczema which will be acceptable to many.

A useful list of the strengths of testing substances is given. The less common varieties of skin cancer, such as the intra-epidermal type, are dealt with at length. The work of Australian dermatologists on the influence of sunlight as an important ætiological factor in the production of skin cancer is recognized. All types of physical therapy are discussed in the chapter on radiant energy.

The concluding chapter, "Miscellaneous" deals with a number of unassociated dermatoses. We note with regret that little progress has been made in the elucidation of the causes of psoriasis.

It is to be hoped that this book will be the forerunner of other editions, as modern research advances so rapidly that individual editions soon become obsolete.

[1] "Recent Advances in Dermatology", by W. N. Goldsmith, M.A., M.D., M.R.C.P., with foreword by A. M. H. Gray, M.D., F.R.C.P., F.R.C.S.; 1936. London: J. and A. Churchill Limited. Demy 8vo, pp. 537, with illustrations. Price: 18s. net.

HYGIENE AND PUBLIC HEALTH.

THE appearance of a new edition of Ghosh's book on hygiene and public health once every three years since it was first published in 1912 is good evidence that it fills a definite niche in the wants of Indian students and medical men. The contents and style of the book confirm this impression. It is a work of great value to medical students in all parts of the world, and there is much in it of interest to the average medical man in Australia. Special stress is of course laid on the conditions of life and the diseases peculiar to a thickly populated tropical country; but at the same time each chapter, while packed with information, is so well arranged and expressed in such lucid English that one is certain to find something of genuine interest at any page that is read. The 172 illustrations are on the whole well selected to supplement the information given in the text, a word of special praise being due to those of the parasites and disease-conveying insects.

A due balance has been preserved in the treatment of the very varied parts of the subject as a whole. For instance, ten pages are given to an interesting discussion on diet in the tropics for natives and Europeans, and an attempt is made to meet the problem of an insufficiency of protein in the case of the vegetarian section of the population. Over one hundred pages are devoted to epidemiology and preventible diseases, the sections on plague, cholera, tuberculosis and leprosy being brief, but omitting nothing of importance. For the most part the findings of recent experts are briefly stated, the brevity in some cases giving perhaps an appearance of dogmatism.

For a medical man who delights in "browsing" the texts of the *Calcutta Municipal Act*, *The Indian Factories Act*, 1934, and a number of official instructions to health officers and inspectors will well repay perusal. This is a good book, turned out in first class style by the publishers.

[1] "A Treatise on Hygiene and Public Health, with Special Reference to the Tropics", by B. N. Ghosh, F.R.F.P. and S., revised and largely rewritten with the assistance of A. D. Stewart, C.I.E., M.B., F.R.C.S.E., D.P.H., D.T.M. and H.; Eighth Edition; 1935. Calcutta: Scientific Publishing Company. Demy 8vo, pp. 675, with illustrations. Price: 12s. 6d. net.

HISTORY OF TUBERCULOSIS: A CORRECTION.

THE name of the publisher was unfortunately omitted from the footnote to the review that appeared in the issue of July 11, 1936, at page 52, of a history of tuberculosis, being number XVI of the series "Clio Medica: A Series of Primers on the History of Medicine". The publishers are Messrs. Paul B. Hoeber, Incorporated, of Fifth Avenue, New York City.

The Medical Journal of Australia

SATURDAY, AUGUST 1, 1936.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

THE ADELAIDE CONGRESS.

At its meeting of September, 1935, the Federal Council of the British Medical Association in Australia accepted the invitation of the South Australian Branch that the fifth session of the Australasian Medical Congress (British Medical Association) should be held in Adelaide. At its meeting of April, 1936, the Federal Council adopted the recommendation of the South Australian Branch that the date of the congress should be August 23 to 28, 1937; these facts have already been noted in this journal. Members of the several Branches should now make their plans for the coming twelve months. Several organizations outside the British Medical Association claim the attention of medical practitioners and certain of them hold annual gatherings at which valuable and important discussions take place. It is well to remember, however, that most of these medical organizations have come into existence almost as offshoots of the British Medical Association. Nor must their members forget—some perhaps do not realize it—that if the larger body is not strong, healthy and flourishing, the smaller groups will eventually languish. The conclusion to be drawn is obvious. The Australasian Medical Congress is held only once in three years. If members of special bodies can during 1937 attend only one central gathering, they should make the Adelaide congress their choice for that year.

The Australasian Medical Congress is, of course, not intended only for those who have interest in special subjects. The main body of the medical profession in Australia consists of general practitioners, and the subjects discussed at sessions of congress have always been those with which the general practitioner is concerned. It is at meetings of congress that he hopes to make personal contact with those who have devoted special study to the subjects on the agenda paper. Previous sessions have been interesting and also successful. The first session, held in Melbourne under the presidency of the late George Adlington Syme, set a high standard; and we think that the standard has been maintained. The second session, it will be remembered, was held at Dunedin, when Sir Louis Barnett was President. Dr. George Abbott was President of the third session in Sydney in 1929; and Dr. D. H. E. Lines presided at the fourth and latest session in Hobart in 1934. The fifth session, with Sir Henry Newland as its President, will, with the earnest cooperation that is already being shown by the South Australian members, be a worthy member of the series.

Members of the Branches in Australia do not need to be reminded of what they may expect to gain by attendance at such a meeting as an Australasian Medical Congress. The memory of the one-hundred-and-third annual meeting of the British Medical Association is still fresh in the minds of the large number who attended it. There is no reason why Australasian gatherings of the Association, though perhaps not so large, should be less enthusiastic or fail to attain such a high scientific standard. Sir Henry Newland has at his back a splendid band of workers. Dr. A. D. Lamphee and Dr. C. B. Sangster are the Joint Honorary Secretaries, Dr. F. S. Poole is the Honorary Treasurer;

and the several committees are at work on the details of the meeting. In the near future notes of the plans and arrangements will be published from time to time in this journal. Members are invited to keep themselves informed of what is being done and to bring the congress to the notice of younger graduates who have not yet formed the congress habit.

In conclusion we should like, on behalf of the medical profession of Australia, to express appreciation both of the generosity of the Chancellor and Council of the University of Adelaide in placing the University buildings at the disposal of the Congress Executive and of the support and cooperation already shown by them.

Current Comment.

PUNCH-DRUNK.

A GOOD deal of interest has been aroused in the curious clinical syndrome known as "punch-drunk" observed in boxers who have been courageous and persistent rather than outstandingly successful, and reference has been made to it recently in this journal. Edward J. Carroll has recently contributed a brief study of this condition in which he has assembled a composite picture derived from many boxers whom he has studied at close quarters.[1] Though some of the not inappropriate synonyms for this condition he quotes may be amusing, such as "slap-happy", and "slug-nutty", the state itself is serious, as it leads to definite organic cerebral changes and well-marked mental reduction. Carroll considers that this species of traumatic encephalopathy tends to be self-limited rather than progressive, though all will not agree with this sweeping statement. The sufferers themselves are pathetically euphoric as regards their capacity, and often continue to engage in matches or to act as training partners, thus exposing themselves to repeated head injuries and therefore to exaggeration of their already damaged state. Carroll's interest has been chiefly psychiatric, and he emphasizes the importance of the temperament of the boxer in determining whether he is likely to suffer from the punch-drunk syndrome. Thus the unimaginative fighter seeks merely to inflict punishment and is prepared to accept it, in this way receiving repeated head injuries, each perhaps more or less minor (especially to the man whose brute strength by far exceeds his sensibilities), but none the less fraught with eventual risk. The clever boxer is able to avoid serious damage, his speed and coordination are of higher degree, and his mentality not only aids him in his campaign, but it enables him to perceive the danger of continuing to pit his waning forces against greater youth and strength. The case of Tunney is cited, who was wise enough to read the writing on the wall when, after a blow received in training, he was completely disorientated for several days.

This curious occupational disease is, of course, of very limited interest in itself, but it presents some difficult problems and some plain warnings. Carroll discusses the question of ætiology, concerning which there is little direct evidence available. He has seen encephalography carried out in two cases; in one instance no changes were found; in the other definite cortical atrophy was present in the frontal region; there was no evidence of arachnoiditis. He remarks further that the absence of local signs in the punch-drunk syndrome makes the existence of arachnoidal changes unlikely. He favours the hypothesis that repeated traumata cause either multiple punctate hæmorrhages or direct cellular damage in the brain, followed by a fine gliosis. A most interesting problem is to explain why such plainly evident objective mental symptoms occur, whereas in the post-concussive states the subjective states predominate. The punch-drunk boxer, of course, shows many other physical phenomena, such as disturbances of reflexes, of coordination and of speech, but in spite also of his obvious lack of judgement and concentration he is quite unaware of the full implications of his condition and has no subjective mental symptoms. The post-concussive patient is painfully aware of his shortcomings; in fact his persistent complaints combined with his lack of objective signs often make him unpopular in out-patient clinics. However, whatever may be the exact pathological changes underlying this remarkable syndrome, it would be wise to take heed of Carroll's statement that: "It is probable that no head blow is taken with impunity, and that each knock-out causes definite and irreparable damage."

[1] *The American Journal of the Medical Sciences*, May, 1936.

EPIDEMIC MYALGIA.

FOR many years there have been occasional epidemics of what appears to be a severe though transient inflammatory disturbance in the trunk muscles, affecting chiefly those of the lower part of the thorax and the upper part of the abdomen. Such cases have been reported from various parts of the world, and such names as "the Bornholm disease" have been applied to local outbreaks, though these do not appear to be essentially different from those seen elsewhere. It is probable that such recurring epidemics of myalgia have been responsible for the constant appearance of the term "pleurodynia" in tables of differential diagnosis of diseases of the chest, and this name is used by M. B. Wellborn in describing a local outbreak in

Cincinatti.[1] The cases he has studied occurred in the personnel of the Cincinatti General Hospital and were typical of many others observed by local practitioners. The chief symptom was an excruciating pain in the chest or epigastrium; this was of sudden onset, was aggravated by movement or deep breathing, and was usually accompanied by tenderness of the affected area. A chill was common, fever was constantly observed and ranged from 100° to 105° F., but there was no increase in the leucocytes, even sometimes a slight decrease. No evidence of pleurisy or pneumonia was observed in this series, nor was there any cough, and recovery was usually prompt and complete, though relapses sometimes occurred and were as a rule severe. The disease attacked young healthy people in the main and was observed during the summer months. There was a vague incubation period during which the patients experienced lassitude, but the acute onset was quite a dramatic event in at least half the cases, the pain attaining a maximum in four to twelve hours. One patient, a doctor, was carefully studied, but no light could be shed on the ætiology. X ray films of the chest were normal, the blood was sterile on culture, and even a slip of the affected *latissimus dorsi* muscle was excised, but also proved sterile on culture and revealed no histological changes.

A similar account of this strange epidemic disease is given in the same journal by F. K. Harder, who is interested in the subject as a public health officer in Cincinatti. He remarks that the Press had described this malady so graphically that the popular name of "devil's grip" was correctly applied by many sufferers who were able to diagnose their own condition without recourse to medical assistance. Harder has collected and summarized previous accounts of American epidemics and has also analysed the Ohio outbreak. Graphs show the steep rise in the incidence curve during the middle of summer, and also that the higher distribution in the younger age groups, up to the age of thirty years. Harder records that in a few instances a friction rub was heard over the affected area of the chest, and, though this physical sign was usually transient in a few cases, it persisted for a time after the subsidence of symptoms. No light was shed upon the ætiology, and the suggestion of Small that a plasmodium was the cause could not be confirmed.

Though of no known importance save in its inconvenience and discomfort, epidemic myalgia may simulate abdominal disease, and operations for appendicitis have actually been performed in such cases. Its confusion with thoracic disease is also easy, especially as true pleurisy does occur. This appears not only from the American reports, but from the cases published in England some years ago, where pleurisy was sufficiently common to warrant the name of "epidemic pleurisy" being applied to the condition then observed. It would be strange if this disease did not occur from time to time in Australia; indeed suggestive cases have from time to time been seen in various centres. Practitioners should therefore be on the look-out and report such cases as do occur.

[1] *The American Journal of the Medical Sciences*, May, 1936.

RESEARCH IN TROPICAL MEDICINE.

In another page in this issue will be found an announcement regarding fellowships and appointments for research in tropical medicine. This announcement represents the first action taken by the Medical Research Council on the advice of the Tropical Medical Research Committee, which they recently appointed after consultation with the Colonial Office. It is considered that the most effective means of promoting investigations in tropical medicine will be the establishment of a small staff of highly qualified workers giving their whole time to research under the best conditions. It is therefore believed that the greatest service which the Council will render by its entry into this field will be the recruitment of able young investigators willing to work in the tropics under a scheme of this kind. For this purpose it has seemed advisable both to offer fellowships for the training of suitable men and to hold out the inducement of permanent and pensionable research posts for those who show special ability.

It will be appreciated that the fellowships which are immediately available will qualify their holders for other appointments than those which will ultimately be made by the Council. There are various opportunities, both in the tropics themselves and in schools of tropical medicine and other institutions, for which men who have had this training should be particularly eligible. In this connexion it may be emphasized that the efforts of the Medical Research Council to recruit men to this field have the support of the Colonial Office and the active cooperation of the schools of tropical medicine in Great Britain.

THE SIR RICHARD STAWELL ORATION.

The Medical Secretary of the Victorian Branch of the British Medical Association announces that the third Sir Richard Stawell Oration will be delivered by Dr. C. T. C. de Crespigny, of Adelaide, at the Medical Society Hall, Albert Street, East Melbourne, on Friday, August 21, 1936. The subject of the oration will be: "The Outlook on Modern Medicine: A Physician's View".

THE AUSTRALIAN AND NEW ZEALAND ASSOCIATION FOR THE ADVANCEMENT OF SCIENCE.

Attention is directed to an announcement appearing on page 168 about the meeting of the Australian and New Zealand Association for the Advancement of Science, to be held at Auckland in January, 1937. Full information may be obtained from the Honorary General Secretary of the Association, Dr. A. B. Walkom, Science House, Sydney.

Abstracts from Current Medical Literature.

OPHTHALMOLOGY.

The Lid-Closure Reflex of the Pupil.

B. BOSHES AND L. L. MAYER (*American Journal of Ophthalmology*, November, 1935) report a case of the lid-closure reflex of the pupil. The right pupil, in a woman of forty-two, was dilated and did not react directly or indirectly to light or accommodation. When the lids were gently closed and then opened, it was noted that the pupil was tiny. The left pupil reacted normally and presented the same phenomenon. Atropine prevented the reflex; the reflex was also prevented by an injection of "Procaine" at the stylomastoid foramen. Adler states that this reaction is usually regarded as proof of a direct connexion between the Edinger-Westphal nucleus and the facial nucleus.

Microscopy of the Retinal Circulation.

R. K. LAMBERT (*American Journal of Ophthalmology*, November, 1935) claims priority in the use of photomicrography in experiments on living animals. He stresses the fact that intraocular vessels acquire the support of the intraocular pressure which eliminates the need for the usual sustaining thickness of the vessel wall. In retinal arteries of 70 micra diameter it is difficult to demonstrate more than one type of cell in the structure of the wall, which usually presents the appearance of endothelium. Adrenaline was administered intravenously in ten experiments on cats, while in each experiment a series of about twenty photographs was taken. The blood pressure was read directly from a manometer in the femoral artery. In six experiments there was measurable vaso-dilatation in the retinal vessels concomitant with the rise in blood pressure. This response is probably due to the absence of muscular tissue from the retinal wall. Amyl nitrite was inhaled by or nitroglycerine was administered intravenously to six cats and photographs were taken. In five animals there was narrowing of the retinal vessels. This finding leads to the belief that the use of amyl nitrite in the treatment of embolic closures of the retinal vessels is physiologically unsound. The cervical sympathetic nerve was repeatedly stimulated and a series of photographs of the retina was taken in fifteen cats and two monkeys. It was not possible to induce any change in the calibre of the retinal arteries by this means.

Subjective Studies of the Blind Spot and Visual Fields.

EDWARD JACKSON (*American Journal of Ophthalmology*, January, 1936) describes how a subject can see his own blind spot. "Closing the eyes when facing the sun, or in bright daylight, or facing at night a 100-watt electric lamp placed within two feet of the eyes, one can perceive two dark spots in the luminous visual field. The first striking characteristic of these spots is that they seem almost constantly to be moving in the field of vision . . . . Each spot always moves in the same direction and to the same extent as its fellow . . . . The colour of the blind spots thus seen is a neutral gray." Within the last year the author has seen two patients who complained of "spots before the eyes", which, judging from their size, shape and position, and the condition under which they were observed, were due to perception of their blind spots. Explanation of the phenomenon satisfied these patients.

Recession Operation.

O. B. NUGENT (*American Journal of Ophthalmology*, August, 1935) performs the recession of the internal rectus tendon in the following manner. A ten-millimetre incision is made vertically over the insertion of the tendon. The capsule is incised above and below, two millimetres behind the incision, and the strabismus hook is inserted; this is followed by insertion of the muscle clamp. This includes conjunctivæ, capsule and muscle. The tendon is now severed from the sclera. A double-armed silk suture is inserted behind the clamp from within outward, merging 1·5 millimetres above and below the centre of the muscle. Each needle is reinserted from without inwards at the upper and lower edge of the muscle. Each needle is then passed through the upper and lower edge of the tendon stump and conjunctivæ and tied loosely pending final adjustment. If the strabismus is over-corrected, the knot is tightened; if it is under-corrected, the capsule is further incised. The long ends of the suture are fastened to the forehead and correction is worn except during sleep.

Treatment of Iris Bombé by Iridectomia ab Externo.

W. B. HORNER (*Archives of Ophthalmology*, January, 1936) advises *iridectomia ab externo* in iris *bombé* when it is difficult or impossible to insert a keratome or Graefe knife. A flap is turned down over the cornea and held with smooth thumb forceps for fixation. The sclera is then scratched one millimetre behind the limbus for seven millimetres with the tip of a keratome. Traction is made by the conjunctival flap and the wound is made to gape. When a perforation is made at one spot a small prolapse of the iris appears. Cuts are continued to each side of the prolapse to the required extent. The incision is enlarged with the tip of the keratome, not with scissors. The prolapsed iris is grasped with forceps, pulled forward and cut with scissors. If no prolapse occurs it signifies peripheral synechiæ and the iris must be separated from the lips of the wound by a spatula, or traction of the flap lifts the edge of its wound and allows easy insertion of forceps with which to pull out a suitable piece of iris. The operation may be used whenever an iridectomy is required. The advantages include its simplicity, freedom from hæmorrhage, good fixation, the fact that there need be no hurry, and the safety of the lens.

Treatment of Sarcoma of the Uveal Tract.

W. G. M. BYERS AND J. A. MACMILLAN (*Archives of Ophthalmology*, December, 1935) discuss the question whether exenteration of the orbit should be done at the outset in every case or whether enucleation should be performed and the treatment governed by subsequent events. In 392 cases of uveal sarcoma extra-ocular extension was found in 113, or 28·83%. So it would seem that exenteration in every case was too radical. If the full length of the optic nerve has not been severed at the enucleation, it should be excised before the wound is closed. For the prevention of general metastasis enucleation is as efficacious as exenteration. After enucleation the tissues should be examined by the pathologist and if extension of the growth to the sclera is demonstrated exenteration should be performed, preferably by electrocoagulation. The value of post-operative radiation is doubtful.

Effect of Bacterial Lysate on Staphylococcic Kerato-Conjunctivitis in Rabbits.

M. M. STRUMIA AND H. W. SCARLETT (*Archives of Ophthalmology*, January, 1936) carried out several experiments to determine the effect of bacterial lysate on the conjunctivitis of rabbits and which portion of the lysate was effective. In a footnote it is stated that the term bacterial lysate is used as synonymous with bacteriophage. The work was suggested by the results obtained with the use of bacterial lysate in the treatment of six patients who were suffering from chronic staphylococcic blepharo-conjunctivitis and one patient with corneal ulcer in both eyes. The cases are shortly described; the condition was prolonged and resisted the usual methods of treatment. An autogenous bacterial lysate from *Staphylococcus aureus* was prepared and used as an ointment with curative result. Conjunctivitis was produced in 62 sensitized rabbits by corneal injection. The effects of the following substances were studied in nine thoroughly controlled series: (i) bacterial lysate containing active lysin (bacteriophage), (ii) heated bacterial lysate not containing active lysin, (iii) bacterial filtrate, (iv) nutrient broth, (v) plain ointment, and (vi) atropine. It was found that the bacterial lysate, the heated bacterial lysate and the bacterial filtrate had the same rapid healing effect. A slight effect was obtained from plain broth, but the others were inert. The action of the bacterial lysate appears

to be due not to the action of the lytic principle itself (bacteriophage), but to the dissolved bacterial proteins, probably through a process of local absorption followed by local and general immunity.

OTO-RHINO-LARYNGOLOGY.

Tuberculosis of the Larynx requiring Tracheotomy.

M. C. MYERSON (*Archives of Otolaryngology*, January, 1936) discusses tuberculosis of the larynx requiring tracheotomy and reports a series of cases. Of 728 cases of laryngeal involvement in a total of 6,516 cases of pulmonary tuberculosis observed during a three-year period tracheotomy was performed in nine. Several patients had been referred to the oto-laryngological service of a large hospital for treatment of tuberculosis because they were hoarse, and on examination they were found to have a non-tuberculous lesion, such as cyst, vascular polypi, paralysis or carcinoma. Not every patient with pulmonary tuberculosis who is hoarse has tuberculosis of the larynx, although this should be suspected. Tracheotomy does not indicate a hopeless prognosis, nor does the presence of laryngeal involvement. Laryngeal involvement diminishes the prospect of recovery, but it does not mean that the patient is doomed. Laryngeal obstruction is invariably caused by a productive lesion. The most frequent cause of laryngeal stenosis is a fixation of the vocal cord in the mid-line or almost in the mid-line, which is caused by fixation of the crico-arytenoid joint. The prognosis is always better in those cases in which the pulmonary lesions show a tendency to recovery and in which the sputum contains few or no tubercle bacilli. The same conditions influence the prospect in a case of tuberculous involvement of the tracheotomy wound. When the tracheal wound comes in contact with a large amount of bacillus-laden sputum, it usually becomes infected. Early and energetic attention to the involved larynx accomplishes much for these sufferers.

Rhinoscleroma.

WILLIAM B. CHAMBERLIN (*Archives of Otolaryngology*, March, 1936) discusses rhinoscleroma and reminds his readers that a disease, whether of plants or animals, at present rarely remains for a long time confined to any one area or locality, but, owing to the increased facilities of travel and transportation, soon finds its way over the entire civilized world, wherever climatic conditions may permit its development. Scleroma, or rhinoscleroma, is no longer confined to a fairly definite locality in south-eastern Europe, but is fairly well distributed over the entire world. The bacillus of Frisch is easily obtained in all cases in practically pure culture. This bacillus is very similar to the bacillus of Friedländer and of Abel. Although typical local lesions can be produced by injecting the organism into animals, it fails to satisfy the postulates of Koch, in that the disease itself cannot be reproduced in animals or in man. The culture of the bacillus of Frisch is not necessary for the absolute diagnosis of the disease. Necessary, however, is the presence of the vacuolated lace or foam cells of Mikulicz and the hyaline bodies of Unna. The mode of transmission or contagion is still unknown, and until this is discovered no definite means of combating the disease will be available. At the present time X radiation or radium in proper dosage offers the best hope of control or cure.

Treatment of Acute Frontal Sinusitis.

TERENCE CAWTHORNE (*The Journal of Laryngology and Otology*, May, 1936), in a paper dealing with the treatment of acute frontal sinusitis, states that there is an increasing tendency to adopt a conservative attitude towards acute frontal sinusitis. The reasons for this are: (i) that a large majority of cases clear up with simple conservative treatment, and (ii) that extensive surgical procedures on the frontal and ethmoidal sinuses in the early stages of acute infection, before protective barriers have been set up, may start a spreading osteomyelitis, meningitis, or sometimes a cavernous sinus thrombosis. Acute frontal sinus suppuration is practically always accompanied by suppuration in the ethmoid cells or antrum, or both, and it is towards these cells that the first therapeutic efforts should be directed. The amount of drainage afforded to the infected frontal sinus will depend upon the degree of obstruction of the fronto-nasal duct due to the anatomical state of the nose, together with the amount of swelling caused by the infected ethmoidal cells and antrum. Should the obstruction be complete, then the pus retained in the frontal sinus may cause a periostitis, usually in relation to the thin floor. This will cause a certain amount of external swelling. Should the obstruction continue to be unrelieved, the pus may spread from the confines of the frontal sinus to the neighbouring regions and, if left to itself, this pus will burst through the thin floor to invade the orbit. If, however, sufficient nasal drainage can be afforded to prevent the retention of pus under pressure in the frontal sinus, then any external swelling will subside and the likelihood of pus bursting the bounds of the frontal sinus is remote. Unless there is definite evidence that pus has escaped from the frontal sinus into the surrounding tissues, conservative measures should be given a thorough trial before any form of external operation is even considered. Such measures should include frequent steam inhalations and antral puncture and lavage, if necessary, daily. The removal of any pus from the antrum is of the greatest importance. In young patients and in those who are unable to tolerate repeated antrum punctures it is permissible to make a limited opening into the antrum through the inferior meatus, which will be sufficient to admit an antral cannula. In addition to these measures the application of shrinking solutions to the middle meatus and turbinate should be used. Protargol 10% is probably the safest medicament, but it may sometimes be necessary to precede this with a pack of 10% cocaine. External heat in the shape of fomentations, electrically heated pads or light baths, may be of great help. Occasionally it is possible to pass a catheter into the frontal sinus and the pus can be removed by lavage or suction. This is found possible only in relatively few cases. Normal drainage may sometimes be assisted further by inward displacement or infraction of the middle turbinate. As a general rule the removal of the anterior end of the middle turbinate is not a good practice, for two reasons: (i) it violates the principle of not opening up tissue spaces in the early stages of acute infection, and (ii) the reactionary swelling of the mucous membrane around the fronto-nasal duct which inevitably follows this procedure tends to obstruct the duct even further, at any rate for a day or two. If after ten days the condition remains stationary, or if during that time there is reason to suppose that pus has spread beyond the confines of the frontal sinus, then some form of external operation will be necessary. Unless the disease has attacked the diploe of the frontal bone or the meninges, it should be limited to a small opening in the floor of the frontal sinus. At a later date, when the acute symptoms have subsided, it is usually necessary to enlarge the fronto-nasal duct and to exenterate any diseased ethmoidal cells. If the disease has invaded the frontal bone or meninges, the amount of bone to be removed will depend upon the extent of the disease. In any case it should include a generous margin of healthy tissue. The author is quite convinced that many, if not most, of the serious complications which occur are the direct result of untimely interference.

Granulocytic Angina.

IRA FRANK (*Archives of Otolaryngology*, March, 1936) reports three cases of granulocytic angina, two of which proved fatal. Granulocytopenia may be caused by a number of agents, among which the drugs of the coal derivatives series, if used continuously, may be included. A sore ulcerated mouth and throat is a frequent result of granulocytopenia. The presence of throat symptoms with the characteristic blood picture of granulocytopenia verifies the diagnosis of granulocytic angina. If there is a history of the continuous use of drugs of the coal tar series, this should suggest a rational cause for the condition.

Special Article.

(Contributed by request.)

VIRUS DISEASES: THE PRESENT POSITION.

THE classical period of medical bacteriology ended approximately with the close of the nineteenth century. The methods for the study of bacteria had been sufficiently developed to allow the isolation of nearly all the important pathogenic bacteria and the important conception of bacterial toxins was well established. Just at the end of the century came the great discoveries in the protozoological sphere, the recognition of the possibility of transmission by insect vectors and the elucidation of malaria. By the end of the War period, in addition to knowledge of the responsible microorganisms, there had been obtained an adequate understanding of the mode of infection and of the more important immunological reactions of most of the bacteria and protozoa pathogenic for man.

In bacteriology the main task of the past twenty years has been to devise methods for the isolation and study of the "left-overs", the agents responsible for those infectious diseases for which no visible microorganism could be incriminated. These are the filtrable viruses, now usually referred to simply as "viruses", and it is the aim of this article to give a brief general account of the nature of these agents and to discuss some of the human virus infections, in the laboratory study of which important recent advances have been made.

The viruses probably comprise a rather heterogeneous collection of agents, but they may be defined as very small microorganisms, capable of passing through filters which hold back bacteria, that is, having an average diameter of 0·3μ or less, and being capable of growth only within living susceptible cells. This last criterion immediately imposes a strict limitation on the available methods of research. Virus diseases can be studied only when living cells susceptible to the virus in question are available. In general, this means that if we are to investigate a human infectious disease due to a virus, we must find a laboratory animal which can be demonstrably infected by the virus. Further, if progress is to be made, the laboratory animal must be obtainable in large numbers and relatively cheaply. The fact that the chimpanzee is susceptible to the common cold has not yet resulted in any significant advance in the understanding or control of that complaint. On the other hand, the discovery that influenza virus will infect mice will probably within a year or two allow us to obtain most of the information we need to interpret the epidemiology of the disease. Actually, of the specifically human diseases due to viruses, an important proportion have remained almost exempt from laboratory investigation, owing to the absence of suitable experimental animals. These include measles, mumps, varicella and the common cold, as well as such diseases, possibly of virus origin, as *encephalitis lethargica*, disseminated sclerosis and Hodgkin's disease.

The Physico-Chemical Nature of a Typical Virus—Vaccinia.

For many reasons vaccinia virus has been the favourite material for theoretical work on the properties of viruses. It has been an object of interest and controversy since Jenner's first publication in 1798, it can be readily preserved, and its activity is easily tested by inoculation on the rabbit skin. In describing the methods used and the results obtained in modern research on the nature of viruses, it will be convenient to use vaccinia virus as an example.

For over a century and a half it has been known that vaccine lymph must contain a disease-producing something which, by inoculation into children or susceptible animals, will produce a great deal more of the same agent. The task of modern workers has been to determine the nature of this disease-producing, self-multiplying active principle. Is it particulate? If so, how big are the particles, what is their shape, how many of them are there, and what is their chemical constitution? These are elementary questions at a purely physico-chemical level, but most of them have been answered only within the last decade.

It is easy to show that the virus is particulate. If an active filtrate is centrifuged at high speed for some time, most of the activity of the virus will be found in the small amount of sediment which is deposited. In order to get an accurate idea of the size of the particles, three methods have been used, all simple in principle, but requiring elaborate apparatus and much care if satisfactory results are to be obtained. The first method is by selective filtration. Collodion membranes of varying degrees of permeability can be prepared. In principle they are sieves in which the holes, instead of being fractions of an inch in diameter, are minute pores whose average diameter ranges from 1μ (one one-thousandth of a millimetre) down to molecular dimensions. If we know the diameter of the smallest holes through which a particle will pass, we have at once an approximate measure of the size of the particle. By this method the average diameter of vaccinia virus particles is estimated to be about 0·15μ. A stained particle of this size can be seen as an incompletely resolved dot under the ordinary oil immersion lens, and Paschen, in 1909, had described dots of about the same size in Giemsa-stained smears of vaccinia as probably representing the virus. With the development of methods of photomicrography by ultra-violet light it became possible to obtain a more definite microscopic image of vaccinia virus particles. Such photographs show that the particles are spherical or very short oval bodies with a more refractile edge. Some of the oval forms show distinct signs of commencing division, indicating that binary fission is the probable method of reproduction. Measurements give the same 0·15μ as the average diameter.

The third method depends on the fact that on centrifugation small bodies sediment less rapidly than large bodies. Centrifugation methods are less accurate than the other two, but give approximately similar values for the particle size of vaccinia virus.

The approximate chemical constitution of vaccinia virus particles has been determined within the last year or two. If a large area of depilated rabbit skin is inoculated by scarification, about one milligramme of virus particles can be obtained from the early lesions by suitable methods. Microchemical methods allow accurate results to be obtained from a few milligrammes of material, and vaccinia virus particles have been shown to be of approximately the same constitution as bacteria, largely protein, with traces of fatty substances, a good deal of carbohydrate, fairly easily lost on repeated washing, and the usual salts.

The numbers of active virus particles in a given virus preparation are determined by some form of animal titration method. Usually rabbits are inoculated intradermally with tenfold dilutions of the original material. If a dilution of 1 in 10^7 produces a small lesion while the 1 in 10^8 dilution is quite inactive, the virus is said to have a titre of 10^7, that is, there are at least 10^7 particles in 0·1 cubic centimetre of the original material. More accurate estimates can be made by using a method in which developing eggs are inoculated. If a dilute suspension of vaccinia is dropped onto the sensitive respiratory membrane of the chick embryo, each particle produces a small focal lesion, the number of which can be used as a measure of the activity of the virus. Incidentally, this method of titration gives perhaps the clearest proof of the particulate character of the viruses.

It will be seen that the vague conception of a filtrable virus current twenty years ago has been replaced by something much more precise. Vaccinia virus is a minute spherical microorganism about 0·15μ in diameter, multi-

plying by binary fission, and very similar in chemical constitution to bacteria.

Vaccinia virus is one of the bigger forms of the group. Of the viruses causing human disease, that of psittacosis is the largest, the typical particles being about 0·3μ in diameter; smallpox virus and those responsible for the various pox diseases of animals and birds are of the same size as vaccinia virus, as also is the virus of *lymphogranuloma inguinale* (climatic or tropical bubo). Influenza, rabies and herpes viruses are smaller (about 0·1μ), those of yellow fever and of one form of human encephalitis (St. Louis) are around 0·03μ in diameter, while that of poliomyelitis is the smallest so far found, the particles, 0·01μ in diameter, being actually smaller than the molecules of certain pure proteins.

Immunity to Viruses.

Some of the virus diseases, measles and yellow fever for example, provide the classical examples of long-lasting immunity. Recovery from an attack of the disease usually confers complete immunity for life. At the other end of the scale we have the common cold, which produces at most only a transient immunity for a few months. Understanding of the immunological reactions of viruses is very far from complete. After infection there appear in the blood antibodies which are capable of inactivating the virus, and the interaction between virus and antibody seems to be essentially similar to bacteria-antibody reactions. In the body we have, however, the complication that virus particles multiply only within cells and that in this situation the circulating antibody cannot affect them. A great deal more work will be necessary before the closely related immunological and epidemiological aspects of virus diseases can be discussed in any general terms. From the point of view of the medical practitioner it will probably be of more interest to discuss four viruses only, those of psittacosis, influenza and poliomyelitis, which are the only common human diseases in Australia which are due to well-studied viruses, and vaccinia, and to indicate what bearing recent laboratory work has or may have on the practical problems they present. These four viruses form a representative sample of the whole group, comprising, as they do, the largest and smallest of the viruses, a highly specific respiratory infection (influenza), a typical neurotropic virus (poliomyelitis), and a good example of human infection derived from an animal reservoir (psittacosis).

Vaccination.

The time-honoured method of preparing vaccine lymph from calves is still practically universal in State and commercial laboratories, but will almost certainly be superseded in a few years by more scientific methods. The great disadvantage of the present method lies in the fact that the raw lymph is heavily contaminated with staphylococci, some potentially pathogenic, and before it can be used it must be slowly sterilized by the action of glycerine in the cold.

There are now two alternative methods available for the rapid production of sterile vaccine virus which is immediately suitable for use in human vaccination. Both make use of the tissues of developing chick embryos. In Goodpasture's method the respiratory membrane of the embryo (the chorio-allantois) is inoculated and the virus-produced lesions are allowed to develop for three or four days. The infected membrane is then removed and provides a rich source of virus free from bacteria. One of the great advantages of the method is that in the event of a sudden enormous demand for vaccination, unlimited stocks could be prepared within a week.

Another more complicated method is to use a tissue culture technique, the virus being grown in a mixture of minced chick embryo tissue and modified Ringer solution. From the practical point of view there seems to be no advantage of this method over that using the chorio-allantoic membrane.

Psittacosis.

The microorganism responsible for psittacosis infection of parrots or man is almost too big to be called a virus. Its standard form consists of minute bodies about 0·3μ in diameter, and under certain conditions much larger bodies, 2μ to 3μ across, may develop. However, like all other viruses, it shows no power of growth in the absence of living susceptible cells. In many respects it appears to be intermediate between the typical viruses, such as vaccinia and the Rickettsias, the minute bacilliform organisms responsible for the various typhus-like diseases. Like the latter, the psittacosis virus can be seen in suitably stained smears, the particles taking a basic stain and being usually situated intracellularly. These blue-staining particles are known as L.C.L. bodies, the initials standing for the names of three investigators who independently observed them in 1930.

Human infection by the virus of psittacosis is a rare accident; normally the virus maintains its existence as an almost harmless parasite of the parrot family. In Australia most of the common parrots and cockatoos are more or les widely infected. Between 20% and 30% of young birds show evidence of present or past infection on *post mortem* examination, though actual symptoms of the infection are very rare in freshly caught birds. These latent infections are liable to flare up when recently caught parrots, and particularly cockatoos, are kept under unsatisfactory conditions. Instead of being confined to the spleen and kidneys, the virus becomes generalized throughout the body and is excreted in the droppings and nasal exudate. The danger to human beings lies chiefly in the ease with which virus in the cage dust may be dispersed into the air by the flutterings of the birds. Inhalation of this dust results in a low grade infection of the lungs, particularly in older people. In general, the symptoms of psittacosis resemble those of typhoid fever. The presence of lung signs indicating an area of partial consolidation, the history of contact with sick parrots or cockatoos, and laboratory examination of sputum for the virus are the main points in establishing the diagnosis.

Although the great majority of human infections are derived from sick parrots, there are fairly numerous instances of infection from birds which showed no obvious symptoms, and also a few instances of human to human infection. In Australia, sick cockatoos seem to be the greatest danger, but aviary-bred budgerigars have been found responsible for two of the cases observed recently in Victoria.

Influenza.

The greatest bacteriological discovery of the past five years has been the demonstration that influenza virus can be transmitted to laboratory animals. Early in 1933 it was shown by Smith Andrewes and Laidlaw that from certain cases of typical influenza a filtrable virus could be obtained which was capable of producing characteristic symptoms in ferrets. In the following year they found that this virus, after a number of passages through ferrets, could produce pneumonia in mice when administered intranasally. Since mice are easily bred in large numbers, their use made it possible to carry out extensive experiments on the nature of the virus and the presence and amount of antibody in human sera. More recently still, work at the Walter and Eliza Hall Institute, Melbourne, has shown that the influenza virus can be adapted to grow and to produce characteristic lesions on the developing egg. For some types of experiment this method is even more suitable than the method of intranasal inoculation in mice.

There is no reason to doubt that the virus which produces these infections in laboratory animals is actually the cause of human influenza. Immunologically identical strains have now been isolated in England, in Porto Rico, Philadelphia, and Alaska, and in Melbourne. In each case the isolation has been from patients attacked in the course of a widespread local incidence of influenza. Attempts to isolate the virus from sporadic infections with symptoms

of the same type as epidemic influenza have been unsuccessful. A great deal of work has still to be done on the question, but it seems highly probable that influenza (which we may now define as a human infection produced by this virus) occurs only in typical acute and widespread epidemics. The sporadic cases and the feverish colds often called influenza are due to some other agent or agents. The work of Dochez and his collaborators tends to suggest that many of them are manifestations of the activity of the virus of the common cold.

The characteristics of the influenza virus may be stated as follows. It is a typical filtrable virus, easily preserved in glycerol-saline in the cold, but inactivated by moderate heating (56° C. for one hour) and composed of particles about 0·1μ in diameter. When first isolated from the human case, it is capable of infecting ferrets, producing symptoms very similar to those of human influenza. After passage from ferret to ferret, the virus will produce mild infections of mice on intranasal inoculation, and by passing the virus from mouse to mouse a high grade of virulence for this species may be developed. The lesions produced in the mouse are confined to the lungs. With a virulent strain instillation of one or two drops into the nose results in death of the mouse after four days. The lungs are found to be deep red in colour and completely consolidated. The readiness with which the virulence of influenza virus can be modified on passage is an important characteristic which probably plays its part in human epidemics. The virus will multiply in the tissue of embryo chicks either in the form of tissue culture or on the chorio-allantoic membrane of the developing egg. Virus grown in either of these ways loses a good deal of its virulence for laboratory animals, and there is a distinct hope that in this way an agent suitable for human vaccination against influenza may be obtained.

After an attack of true influenza in man or in the ferret antibodies appear in the serum which can be detected and quantitatively titrated either by mouse or egg titration methods. As would be expected, a large majority of human sera show the presence of such antibodies, but the significance of these antibodies is not clear. Only a small number of tests have as yet been reported in the literature, but, judging from these and from my own limited experience, it seems likely that only persons who at the time have a low antibody content will contract true influenza during an epidemic, but that some persons with little or no antibody have nevertheless a high resistance to infection. It is not unlikely that individuals with a moderately high antibody content may be infected with the influenza virus with symptoms similar to those of an ordinary cold. Tests of serum for the presence of influenza virus antibodies should in time allow us to define clearly what types of respiratory infection are actually due to this virus. It is already clear that true influenza is not so common as is usually supposed. In England many deaths from influenza are registered each year, but the Hampstead investigators found that, while they could isolate the virus in 1932-1933 and 1934-1935, no positive results were obtained in 1933-1934 or 1935-1936. In Melbourne this year there was in the autumn a considerable prevalence of clinical influenza, but none of the patients we examined showed any serological evidence of infection by the virus.

It is highly probable that methods will shortly be available which will allow immunization of human beings against true epidemic influenza of classical type. Whether such immunization will ever be capable of preventing influenza epidemics is more than doubtful. The preparation of influenza virus vaccines will undoubtedly be much more expensive than that of bacterial vaccines, and in order to control the effectiveness of the inoculations serum tests will probably be required—another expensive procedure. An epidemic of influenza appears suddenly and rarely lasts more than six or eight weeks in one place. Immunization at the time of an epidemic will therefore be futile; probably an annually repeated course of inoculations would be required. This may protect the subject against true influenza but will have no influence on the various sporadic maladies that feel like and are called influenza. Finally, we have to realize that the really important and dangerous influenzas, such as the 1918-1919 pandemic, are probably caused by virus strains of serological character different from those of the common strains. This is what makes them so widespread and deadly and, incidentally, makes it impossible to prepare beforehand any immunizing material. On the whole it seems likely that immunization against influenza will never be more than a luxury precaution availed of by a small proportion of well-to-do, intelligent or hypochondriac individuals. Possibly future research will discover a method of natural epidemic immunization by some highly contagious but symptomless strain which will pass naturally through the community and leave it immune to the ordinary symptom-producing strains; but at the present time this is mere fantasy.

Poliomyelitis.

Of recent years there has been a relative diminution in the activity of experimental research on poliomyelitis, but from about 1911 to 1932 it was intensively studied, particularly in America. More money was probably made available for research on infantile paralysis than on any other specific problem in the field of infectious diseases. In the opinion of most competent critics, this work has given us a fairly precise knowledge of the responsible virus and of the pathology and epidemiology of the disease, but it has provided nothing of practical value to the clinician. Unless some entirely new aspect is opened up in the future, it seems most unlikely that infantile paralysis will ever be amenable to public health control or individual prophylaxis.

The disease is due to one of the smallest of the viruses, whose particles are approximately 0·01μ in diameter—one one-hundredth of the diameter of a staphylococcus, one one-millionth of its mass. The virus can be transferred to monkeys and produces, after a few passages, a clinical condition almost identical with a severe attack of infantile paralysis in a child. This susceptibility of the monkey has provided a means of studying the pathology of the experimental disease and of determining whether antibodies were present in human sera. As a result of a great deal of research there is now a reasonable uniformity of opinion amongst laboratory workers in the virus field in regard to the nature of the infective process in poliomyelitis. The following bald summary of the position would probably be regarded as representing a fair approach to the truth by most British and American workers. It is, however, developed largely from analogy with the experimental disease, and it is possible that conditions in man are more different from those in the monkey than is usually believed.

The virus is far commoner than the proportion of typical cases would indicate, and in a city population probably every child is at some time or other exposed to and infected by the virus. More than 99% of these infections are non-paralytic; many are probably completely symptomless. The pathology of these "subclinical" infections is unknown, but there are suggestions that the virus multiplies in the cells of the olfactory system without penetrating further into the central nervous system. In a very small proportion of infected children the virus passes beyond the olfactory system, and probably by spread along the axones of the central nervous system reaches the anterior horn cells of the spinal cord. Here the virus multiplies actively and produces its characteristic lesions. Spread of the virus through the community is probably almost entirely due to persons with asymptomatic infections. The virus is liberated from the olfactory cells and passes into the naso-pharynx, to be discharged into the environment carried on droplets of saliva.

Many attempts have been made to apply the available knowledge concerning the virus and its immunological reactions to the clinical problem of preventing paralytic infection. These attempts have followed one of two lines.

The first involves the treatment of "preparalytic" cases with human convalescent serum. This has been widely

practised, and great success has been claimed by the advocates of the method. When it was first used, treatment by convalescent serum was believed to have a sound experimental basis, the nervous involvement being supposed to be a secondary development following a general systemic infection by the virus. Administration of adequate antibody during the primary stage should, on this view of the pathology of the infection, prevent the occurrence of the secondary nervous involvement. Present-day views, however, regard the virus as a strictly neurotropic one. The infection is throughout confined to the actual nervous tissues and cannot be reached by antibody in any concentration which would be likely to be effective. Theoretical considerations of this sort led to a series of controlled tests on the efficacy of convalescent serum being made in America. None of the five such tests which have been reported showed any benefit from the administration of serum.

More recently attempts have been made to develop methods of active immunization by using monkey virus sterilized by formalin or some other antiseptic. The difficulty here is that completely killed virus appears to be non-antigenic and that only preparations containing some proportion of living virus particles can be effective immunizing agents. The danger of administering such material to children is obvious, and it was not long after large scale immunization had been commenced in America that accidents began to be reported. There seems to be little doubt that administration of such vaccines has been responsible for about a dozen cases of infantile paralysis. It is very unlikely that work on these lines will be continued.

Conclusion.

It will be seen that although our knowledge of virus diseases has been immensely increased in the last few years, we are as far as ever from being able to control those infections of which man is the primary host and which are spread via the respiratory route. Two of these, influenza and poliomyelitis, have been discussed; measles, mumps and varicella fall into the same category. The viruses responsible for these infections form part of the biological environment of the human species and can probably never be eliminated. But it will be noticed that the average severity of the disease induced by any of them in a previously healthy individual is quite low. Raising of the standard of nutrition and hygiene are more likely to influence the death rates from these infections than any specifically immunological measures.

Psittacosis is an example of another group of diseases in which man is not a normal host of the virus. Bubonic plague, though not due to a virus, is biologically similar. In these cases we have only to prevent effective contact between the natural host, parrot or rat, and man in order to eliminate the disease.

F. M. BURNET.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held at the Victorian Eye and Ear Hospital, Melbourne, on June 17, 1936. The meeting took the form of a series of clinical demonstrations by the members of the ophthalmic section of the honorary medical staff of the hospital.

Common External Conditions of the Eye.

DR. MARK GARDNER, DR. L. DUNCAN and DR. STUART FRASER showed a series of patients illustrating the more common diseases of the eyelids, lachrymal apparatus and conjunctivæ. These also included simple catarrhal conjunctivitis, angular conjunctivitis, muco-purulent conjunctivitis, phlyctenular conjunctivitis and trachoma, pterygia, Meibomian cysts and chronic dacryocystitis. In each instance a brief account was given of the diagnosis and treatment, and Dr. Gardner demonstrated the surgical treatment of a Meibomian cyst and a pterygium.

Diseases of the Cornea.

DR. J. A. O'BRIEN and DR. ESME ANDERSON demonstrated a number of patients with diseases of the cornea, including corneal ulceration, interstitial keratitis and *keratitis profunda*. Dr. O'Brien gave an interesting account of the treatment of corneal ulcers and showed one case of dendriform keratitis which had responded to treatment with *Grenz* rays.

Intraocular Lesions.

DR. A. S. ANDERSON, DR. M. YUILLE and DR. R. STOTT demonstrated a number of intraocular lesions, including thrombosis of the central retinal vein, diabetic, renal and arteriosclerotic retinitis, vitreous opacities, and one patient with a piece of steel embedded in the substance of the lens of one eye. Dr. Anderson also showed a child with an enlarged left pupil which responded neither to light nor to accommodation, but contracted after instillation of eserine. Examination of the fundus revealed two small areas of old chorioiditis, and the Wassermann reaction was positive.

Orthoptic Treatment.

Dr. Anderson also arranged a display of the work of the Orthoptic Clinic under the direction of Misses Fox and Bryant, who demonstrated the principles underlying the orthoptic treatment of squint. These were illustrated by two cases, one of divergent strabismus and the other of convergent strabismus, which had derived benefit from this form of treatment.

Radiography.

DR. JOHN O'SULLIVAN showed two cases of epithelioma of the limbus region treated by radon needling. He also demonstrated films of intracranial tumours. Several cases of pituitary tumours were shown and a case of a tumour of the eighth nerve, giving rise to changes in the region of the internal auditory meatus, was demonstrated.

The films of a case of hydrocephalus, showing enlargement of the pituitary fossa and both internal auditory meati, were shown; the condition was secondary to a tumour of the cerebellum. Radiographs of the orbit, showing involvement of the optic foramen by a tumour, were also demonstrated.

DR. CHAS. E. DENNIS showed a number of films demonstrating a series of cerebral tumours, including an hæmangiomatous tumour of the frontal region with irregular hyperostosis of the frontal bone. At the operation the hæmorrhage had been so severe and general when an attempt was made to raise the bony flap that the operation had had to be abandoned. Another film was of an intrasellar pituitary tumour with definite concentric enlargement of the pituitary fossa and partial absorption of the *dorsum sellæ*. Dr. Dennis also showed a large suprasellar cranio-pharyngioma with tremendous enlargement of the *sella turcica* and destruction of the anterior clinoid processes and *dorsum sellæ*, and areas of calcification in and above the sellar region. This tumour had been removed by Dr. Trumble at the Alfred Hospital with great improvement and relief of symptoms. Other films showed the early calcification of both lachrymal glands and a very large, slightly nodular ivory osteoma of the right frontal sinus, apparently arising from the lesser wing of the sphenoid bone.

Dr. Dennis also showed a series of films demonstrating paranasal sinusitis and mastoiditis, and films of three patients, each of whom had a small spicule of bone lodged in the œsophagus. The non-opaque foreign bodies were demonstrated by causing the patient to swallow a spoonful of opaque barium mixture into which cotton wool was shredded; the cotton wool, containing the opaque barium meal, lodged on the foreign body and rendered the presence of the foreign body obvious after the rest of the opaque material had passed on out of the œsophagus.

NOMINATIONS AND ELECTIONS.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Holland, Esme Hope, M.B., B.S., 1932 (Univ. Sydney), 70, Liverpool Road, Summer Hill.

Medical Societies.

THE PUBLIC MEDICAL OFFICERS' ASSOCIATION OF VICTORIA.

THE first annual general meeting of the Public Medical Officers' Association of Victoria was held in the Medical Society Hall, Albert Street, East Melbourne, on May 21, 1936. DR. C. P. ROWAN, the President, was in the chair, and twelve members were present. Apologies were received from eleven others who were unable to be present.

Annual Report.

The annual report read at the meeting showed that much organizing work had been done during the year and that 53 members had been enrolled.

Early in the year the representations to the Premier regarding restoration of salary cuts had been ineffectual.

The Committee had viewed with much apprehension the proposal submitted by the Director for a Board of Control for the Mental Hygiene Department. They conferred with the Council of the Victorian Branch of the British Medical Association, which concurred in their views. A deputation from the Council waited on the Minister for Health and the Director of Mental Hygiene and discussed this matter. The Minister promised to submit the Council's views to Cabinet, and it was agreed that the personnel of any proposed board should be predominantly medical.

The Committee met seven times during the year, attendance of members being as follows: Dr. Rowan, 7; Dr. Beveridge, 7; Dr. Greig, 7; Dr. Johnson, 6; Dr. Dale, 6; Dr. McKay, 2; Dr. Allen, 2; Dr. Adey, 1.

Statements of the existing rates of pay for medical officers in various services in the Australian States showed that the salaries in Victoria were generally much lower than those for similar work done in the other States.

Rules.

Some slight alterations were made to the rules of the Association, one being to determine that no member could hold office as president for two consecutive years.

Election of Office-Bearers.

The following officers were elected for the year: President, Dr. Adey; Honorary Secretary, Dr. Rowan; Honorary Treasurer, Dr. Jago; Honorary Auditor, Dr. Melville; Committee, Dr. Adey, Dr. Jago, Dr. Johnson, Dr. Greig, Dr. Allen, Dr. Beveridge and Dr. Yule.

Salaries.

The members present strongly urged that there should be an early general meeting to discuss the inadequate salaries paid to medical officers in Victoria.

Congresses.

THE AUSTRALIAN AND NEW ZEALAND ASSOCIATION FOR THE ADVANCEMENT OF SCIENCE.

THE programme of addresses and papers for the meetings of the Section of Medical Science and National Health in connexion with the Auckland meeting of the Australian and New Zealand Association for the Advancement of Science, to be held in January, 1937, is now being compiled. The Secretary of the Section, Dr. J. E. Caughey, of Auckland, advises that the following subjects and speakers have already been arranged:

Presidential address—"Heart Disease: An Important Public Health Problem", Dr. A. R. Southwood.

"Hydatid Disease: Research and Prevention", Sir Louis Barnett.

"Characteristics of the New Zealand Climate", Dr. Kidson.

"The Anatomy of the Bronchial Tree", Dr. Hardie Neil.

"Malaria in Melanesia", Dr. Sayers.

"The Social Aspect of Injury", Dr. Selwyn Morris.

Arrangements are also being made for a discussion on national health insurance. It is probable that Professor C. E. Hercus, of Dunedin, who has taken a leading part in medical politics for some years, will contribute a paper on this subject, and it is desired to arrange for a prominent medical man in Australia to speak at the same session.

Nutrition problems will also receive consideration. Dr. E. B. Gunson, of Auckland, will speak on "A National Food Policy Coordinating Agricultural and Health Interests". The discussion on this subject will probably be one of the special features of the section's meetings.

At the present time official medicine—the interrelation of the practising profession and public health authorities and the various aspects of community health—are prominently under discussion, and the consideration given to these matters at the meeting should be extremely useful.

Medical practitioners and others interested who are planning to attend the meeting and who desire to contribute papers at the meetings of the Section of Medical Science and National Health, are asked to communicate with the President of the section, Dr. A. R. Southwood, 170, North Terrace, Adelaide, or the Secretary, Dr. J. E. Caughey, 3, Alfred Street, Auckland.

Research.

FELLOWSHIPS AND APPOINTMENTS FOR RESEARCH IN TROPICAL MEDICINE.

THE Medical Research Council of Great Britain, advised by the Tropical Medical Research Committee which it has recently appointed after consultation with the Colonial Office, announces the following fellowships and appointments.

Junior Fellowships.

Three junior fellowships are offered immediately for award to qualified medical men wishing to receive training with a view to careers in research work in tropical medicine. Preference will be given to candidates who have already had preliminary experience of methods of research in some branch of medical science.

Subject to satisfactory reports, the fellowships will be tenable for three years. The first year will be spent at a school of tropical medicine; the second in doing research in the same or some other institution at home; and the third largely in work under direction at some centre in the tropics. The stipend will be at the rates of £300, £400 and £500 *per annum* in the successive years, with an additional allowance during service abroad and necessary expenses.

The Council has every reason to believe that men who undergo this training will be eligible and well qualified for various appointments, apart from the prospects of further employment under the scheme now announced.

Senior Fellowships.

In three years' time at least one senior fellowship will be available for candidates who have held the junior fellow-

ships mentioned above. This will be awarded for a further period of three years, carrying stipend at the rate of £600 to £750 *per annum*, with an additional allowance during service abroad and expenses. The time will be spent mainly in research work in the tropics.

The Council is also prepared to consider immediate applications for senior fellowships from candidates who have had adequate experience in research work, whether already specially trained in tropical medicine or not.

Permanent Appointments.

The Council further intends to establish in due course, as suitable investigators become available as the result of the fellowships scheme, permanent and pensionable appointments for research work in tropical medicine, including senior posts. Members of this research staff will work partly in the tropics and partly in institutions at home, to which they will be severally attached. The exact terms of service are still undecided, but they will be not less favourable than those which apply to other government appointments at home or overseas for men of similar professional standing.

Inquiries may be addressed to the Secretary, Tropical Medical Research Committee, 38, Old Queen Street, London, S.W.1, with whom applications should be lodged not later than October 1, 1936.

Correspondence.

THE LAW OF THE SPLIT PELVIS.

SIR: I am quite unable to accept the restatement of the law of the split pelvis made by Professor Wood Jones in this journal on June 20, as I consider that it is not consistent with the facts.

He says: "When an animal supports its body weight upon its hind limbs at right angles to the body axis the ventral pelvic symphysis will be elongated." The bipedal birds have the body axis and the hind limbs at right angles, but there is no ventral symphysis, as the pelvis is open—a characteristic avian feature—and in a typical bird the weight is carried entirely on the hind limbs. Again in the reptiles, there is no symphysis in the quadrupedal stegosaurian and ceratopsian members of the Ornithischia (the open pelvis group of extinct dinosaurs), and a similar absence of the ventral symphysis is found in the Soricidæ and Talpidæ amongst the quadrupedal mammals.

Professor Wood Jones also states: "This elongation will diminish as the weight is carried on the hind limbs turned progressively into line with the long axis of the body." This again is not in accord with the facts. The bipedal Ornithischia possessed an open pelvis, but their colleagues, the even more erect bipedal therapods of the saurischian dinosaurs, had a closed pelvis with a characteristic elongated pubic symphysis as well as a smaller ischiatic one. Amongst the mammals in the more or less upright Macropodidæ (kangaroos and wallabies) there is a long ischiopubic symphysis, which is reinforced by a prominent hypoischium. Again, in the semi-erect chimpanzee the symphysis is elongated and ischio-pubic, as it is in the majority of quadrupedal mammals, such as ungulates, carnivores, monkeys *et cetera*.

The Professor concludes his restatement with: "and the symphysis will tend to disappear altogether if the animal ceases to carry its weight on its hind limbs". The plesiosaurs, ichthyosaurs and mososaurs were extinct aquatic reptiles and carried no weight on their hind limbs, but they had prominent pubic and ischiatic symphyses in the pelvis. In the monosaurs and later ichthyosaurs the pelvis lost connexion with the spinal column, as in the Sirenia (dugong, monatu) and Cetacea (whales, porpoises *et cetera*), but the ventral symphysis remained; the later Ichthyosauria lost the pubo-ischiatic opening and the pelvis reverted to the ancestral plate-like condition with its pubo-ischiatic symphysis. Even in the Cetacea the paired degenerate pelvic bones (? ischia) are connected by ligament, representing the vestigial remnant of the symphysis.

In conclusion, I state the law of the split pelvis as before: When disproportion occurs between the products of conception and the pelvic brim, separation of the ventral symphysis occurs in those types which survive.

Yours, etc.,

C. D. GILLIES.

Ridge Street,
Northgate,
Brisbane,
Queensland,
July 7, 1936.

Obituary.

DANIEL FLORANCE MacGILLICUDDY.

WE regret to announce the death of Dr. Daniel Florance MacGillicuddy, which occurred on July 13, 1936, at Richmond, Victoria.

JOHN REGINALD DAVIS.

WE regret to announce the death of Dr. John Reginald Davis, which occurred on July 24, 1936, at Westbury, Tasmania.

JOHN SMITH PURDY.

WE regret to announce the death of Dr. John Smith Purdy, which occurred on July 26, 1936, at Sydney, New South Wales.

Corrigendum.

AN error has occurred in the paper by Dr. P. A. Earnshaw, entitled "Dental Decay as an Index of Malnutrition", published in the issue of July 4, 1936. At page 6 the sixth and seventh lines should read: "Cheese, 1 ounce. Butter, 2 ounces", instead of "Cheese, 2 ounces. Butter, 3 ounces".

NOTICE.

PRIMARY FELLOWSHIP EXAMINATION OF THE ROYAL COLLEGE OF SURGEONS OF ENGLAND.

THE Secretary of the Royal Australasian College of Surgeons announces that a primary examination in anatomy and physiology for the Fellowship of the Royal College of Surgeons of England will be held in Melbourne and Sydney on Monday, November 29, 1937, and following days. Copies of the regulations, schedules of the required certificates, and further particulars may be obtained from the Secretary of the Royal Australasian College of Surgeons, Spring Street, Melbourne.

AUSTRALIAN ARMY MEDICAL CORPS OFFICERS' MESS, SECOND MILITARY DISTRICT.

THE annual general meeting of the Australian Army Medical Corps Officers' Mess, Second Military District, will be held at the Small Arms School, Canberra Street, Randwick, at 8 p.m. on August 8, 1936. Following the business of the meeting the Committee has arranged for the exhibition of some interesting films which are now being used in the training of various units. Supper will be served afterwards. A cordial invitation is extended to officers who formerly served with medical units or who are now on the Reserve or Retired Lists.

Books Received.

THE MOTHER'S ENCYCLOPÆDIA (British edition), edited by L. Chaloner; 1936. London: George Allen and Unwin Limited. Demy 8vo, pp. 692, with illustrations. Price: 8s. 6d. net.

A HANDBOOK OF TROPICAL THERAPEUTICS, by R. N. Chopra, C.I.E., K.H.P., M.A., M.D., M.R.C.P.; 1936. Calcutta: Art Press. Demy 8vo, pp. 1769. Price: Rs. 25.

THE TOXÆMIAS OF PREGNANCY, by L. McIlroy, D.B.E., LL.D., M.D., D.Sc., F.C.O.G., M.R.C.P.; 1936. London: Edward Arnold and Company. Demy 8vo, pp. 366. Price: 14s. net.

Diary for the Month.

AUG. 3.—New South Wales Branch, B.M.A.: Organization and Science Committee.
AUG. 4.—Tasmanian Branch, B.M.A.: Council.
AUG. 5.—Western Australian Branch, B.M.A.: Council.
AUG. 5.—Victorian Branch, B.M.A.: Branch.
AUG. 6.—South Australian Branch, B.M.A.: Council.
AUG. 7.—Queensland Branch, B.M.A.: Branch.
AUG. 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
AUG. 11.—Tasmanian Branch, B.M.A.: Branch.
AUG. 14.—Queensland Branch, B.M.A.: Council.
AUG. 18.—New South Wales Branch, B.M.A.: Ethics Committee.
AUG. 18.—Tasmanian Branch, B.M.A.: Council.
AUG. 19.—Western Australian Branch, B.M.A.: Branch.
AUG. 20.—New South Wales Branch, B.M.A.: Clinical meeting.
AUG. 24.—Federal Council.
AUG. 25.—New South Wales Branch, B.M.A.: Medical Politics Committee.

Medical Appointments.

Dr. F. H. H. Wilson has been appointed Deputy Medical Superintendent, Prince Henry Hospital, Office of the Director-General of Public Health, New South Wales.

* * *

Dr. T. R. Gaha and Dr. A. B. Cunningham have been appointed members of the Board of Optical Registration, Tasmania, pursuant to the provisions of the *Opticians Act*, 1913.

* * *

The undermentioned have been appointed members of the Medical Board of South Australia: Dr. L. W. Jeffries, Dr. E. A. Johnson, Dr. R. S. Rogers, Dr. H. H. E. Russell and Dr. A. M. Cudmore.

* * *

The undermentioned appointments have been made at the Adelaide Hospital, South Australia: Dr. G. H. Burnell, Honorary Consulting Adviser to the Night Clinic (male section); Dr. R. F. Matters, Honorary Consulting Adviser to the Night Clinic (female section).

* * *

Dr. J. R. Shannon has been appointed Assistant Medical Officer of Health, Office of the Director-General of Public Health, New South Wales.

* * *

Dr. C. W. England has been appointed Medical Officer, Office of the Director-General of Public Health, New South Wales.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xxii-xxiv.

HORNSBY AND DISTRICT HOSPITAL, HORNSBY, NEW SOUTH WALES: Honorary Officers.

METROPOLITAN INFECTIOUS DISEASES HOSPITAL BOARD, ADELAIDE, SOUTH AUSTRALIA: Junior Resident Medical Officer.

THE UNIVERSITY OF SYDNEY, NEW SOUTH WALES: Chair of Psychiatry.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY Hospital are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

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